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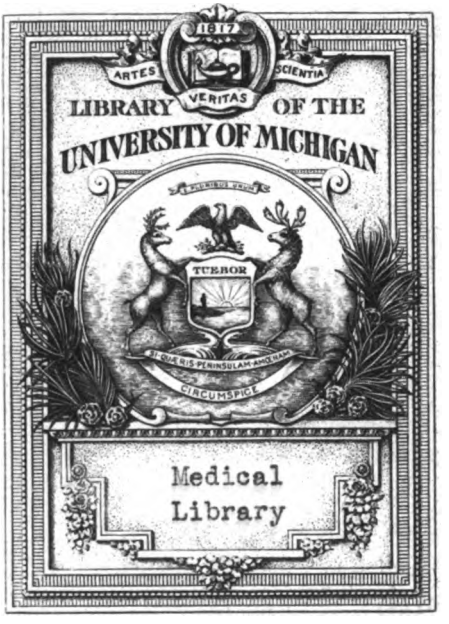

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TRANSACTIONS OF THE

SECTION ON

Diseases of Children

of the
American Medical Association
at the Sixty-Fourth Annual
Session, held at Minneapolis,
Minn., June 17 to 20, 1913

AMERICAN MEDICAL ASSOCIATION PRESS
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PROCEEDINGS OF THE SECTION

TUESDAY, JUNE 17—AFTERNOON

The meeting was called to order by the Chairman, Dr. Henry Dwight Chapin, New York, at 2:20 p. m.

Dr. Chapin read the Chairman's address, entitled "Radiographic Studies of the Intestines in Infants."

Dr. John Lovett Morse, Boston, Mass., read a paper entitled "The Use of the Roentgen Ray in the Diagnosis of Obscure Abdominal Conditions in Infants and Children."

Dr. L. R. DeBuys, New Orleans, read a paper entitled "The Use of the Roentgen Ray in Pyloric Obstruction."

These papers were discussed by Drs. Abraham Jacobi, New York; G. R. Pisek, New York; Rufus Cole, New York; A. W. Crane, Kalamazoo, Mich.; Henry Helmholz, Chicago; H. Lowenburg, Philadelphia; John Lovett Morse, Boston; L. R. De Buys, New Orleans, and Henry D. Chapin, New York.

Dr. A. L. Goodman, New York, read a paper entitled "Rupture of Mediastinal Lymph-Node into Bronchus." Discussed by Drs. Willy Meyer, New York; I. A. Abt, Chicago; A. W. Myers, Milwaukee, Wis.; C. F. Wahrer, Fort Madison, Iowa, and A. L. Goodman, New York.

Dr. Frank Spooner Churchill, Chicago, read a paper entitled "Report of a Case of Staphylococcus Meningitis in a Child." Discussed by Drs. H. M. McClanahan, Omaha; H. Lowenburg, Philadelphia; F. P. Gengenbach, Denver, and Frank S. Churchill, Chicago.

WEDNESDAY, JUNE 18—MORNING

The first paper was by Dr. Thomas C. McCleave, Berkeley, Cal., entitled "Acute Acid Intoxication in Children." Discussed by Drs. John Zahorsky, St. Louis; J. R. Snyder, Birmingham, Ala.; C. H. Johnston, Grand Rapids, Mich.; Granville D. Ryan, Des Moines, Iowa; Thomas B. Cooley, Detroit; I. A. Abt, Chicago; J. L. Morse, Boston; H. T. Price, Pittsburgh, Pa.; L. R. DeBuys, New Orleans; H. Lowenburg, Philadelphia, and T. C. McCleave, Berkeley, Cal.

Dr. Victor C. Vaughan, Ann Arbor, Mich., read a paper entitled "The Protein Poison and Its Relation to Disease." Discussed by Drs. John Zahorsky, St. Louis; Mary Strong, Omaha; C. H. Johnson, Grand Rapids; C. G. Grulee, Chicago; Louis Burckhardt, Indianapolis; C. F. Wahrer, Fort Madison, Iowa; H. W. Cheney, Chicago; George S. Cattermole, Boulder,

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Colo.; T. C. McCleave, Berkeley, Cal., and Victor C. Vaughan, Ann Arbor, Mich.

Dr. Clifford G. Grulee, Chicago, read a paper entitled "The Nature of Ileocolitis from an Etiologic Standpoint." Discussed by Drs. R. M. Smith, Boston; E. W. Mitchell, Cincinnati; J. L. Morse, Boston, and Clifford G. Grulee, Chicago.

Dr. I. A. Abt, Chicago, read a paper entitled "Nutritional Injuries Produced by Starch." Discussed by Drs. John Zahorsky, St. Louis; E. P. Gengenbach, Denver; F. W. Schultz, Minneapolis; Julius Hess, Chicago; L. D. Buckley, New York, and I. A. Abt, Chicago.

Wednesday, June 18—Afternoon

Dr. Walter L. Bierring, Des Moines, Iowa, read a paper by himself and Drs. V. L. Glomset and J. A. Goodrich, Des Moines, Iowa. Discussed by Drs. C. G. Grulee, Chicago; C. F. Wahrer, Fort Madison, Iowa; Mary Strong, Omaha; John Zahorsky, St. Louis, and W. L. Bierring, Des Moines, Iowa.

Dr. Thomas B. Cooley, Detroit, read a paper entitled "The Treatment of Hemorrhagic Conditions in Infancy and Childhood." Discussed by Drs. V. D. Lespinasse, Chicago; H. M. McClanahan, Omaha; Julius Hess, Chicago; Ogden M. Edwards, Pittsburgh; H. Lowenburg, Philadelphia; C. Brooks, Pittsburgh, Pa., and T. B. Cooley, Detroit.

Dr. Arthur E. Hertzler, Kansas City, Mo., read a paper entitled "Treatment of Inguinal Hernia in Children." Discussed by Dr. C. G. Buford, Chicago.

Dr. William Cavan Woolsey, Brooklyn, read a paper entitled "General Anesthesia in the Surgery of Childhood." No discussion.

Dr. Julius Parker Sedgwick, Minneapolis, read a paper entitled "Spasmophilia with Especial Reference to Familial Reactions and Repeated Absences." Discussed by Drs. A. C. Rogers, Faribault, Minn.; Mary Strong, Omaha, and John Zahorsky, St. Louis.

Dr. A. C. Rogers, Faribault, Minn., read a paper entitled "Desirability of Early Diagnosis of Mental Deficiency." Discussed by Drs. Mary Strong, Omaha; C. F. Wahrer, Fort Madison, Iowa, and A. C. Rogers, Faribault, Minn.

Thursday, June 19—Morning

The following officers were elected: Chairman, Dr. F. S. Churchill, Chicago; Vice-Chairman, Dr. L. R. DeBuys, New Orleans; Secretary, Dr. F. P. Gengenbach, Denver; Delegate, Dr. J. P. Sedgwick, Minneapolis.

Dr. Richard M. Smith, Boston, read a paper entitled "Vulvovaginitis in Children." Discussed by Drs. F. C. Neff, Kansas City, Mo.; H. M. McClanahan, Omaha; H. Lowenburg, Phila-

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delphia; D. L. Richardson, Providence, R. I.; J. L. Hess, Chicago; C. F. Wahrer, Fort Madison, Iowa; John Zahorsky, St. Louis; H. D. Chapin, New York, and R. M. Smith, Boston.

Dr. John Zahorsky, St. Louis, read a paper entitled "Roseola Infantum." Discussed by Drs. F. P. Gengenbach, Denver; H. W. Cheney, Chicago, and John Zahorsky, St. Louis.

Dr. A. Sophian, Kansas City, Mo., read a paper entitled "Atypical Forms of Meningitis (Posterior Basic Meningitis; Aseptic Meningitis)." Discussed by Drs. J. L. Morse, Boston, and A. Sophian, Kansas City, Mo.

Dr. Julius H. Hess, Chicago, read a paper entitled "Leukocyte Counts in Epidemic Meningitis and Pneumonia." No discussion.

Dr. F. P. Gengenbach, Denver, read a paper entitled "Precocious Menstruation." No discussion.

Dr. H. M. McClanahan, Omaha, read a paper entitled "Clinical Report and Post-Mortem Findings in the Case of a Child with a Congenital Heart Disease, Dying from Acute Nephritis." Discussed by Drs. John Zahorsky, St. Louis; C. F. Wahrer, Fort Madison, Iowa; H. Lowenburg, Philadelphia, and H. M. McClanahan, Omaha.

Thursday, June 19—Afternoon

Dr. D. L. Richardson, Providence, R. I., read a paper entitled "The Hospital Management of Contagious Diseases." Discussed by Dr. H. E. Roberts, Minneapolis.

Miss Julia C. Lathrop, Washington, D. C., read a paper entitled "Federal Children's Bureau; the Law, the Organization, the Scope of Its Present Work." Discussed by Drs. C. F. Wahrer, Fort Madison, Iowa, and H. D. Chapin, New York.

Dr. H. Lowenburg, Philadelphia, read a paper entitled "The Etiology of Artificial Feeding; a Plea for the Study of Breast Milk Problems." Discussed by Drs. Rosenthal Edmondson, Grand Rapids, Mich.; John Zahorsky, St. Louis; C. F. Wahrer, Fort Madison, Iowa; C. F. Chapin, New York; H. W. Bancroft, Bancroft, Neb.; L. R. DeBuys, New Orleans; C. H. Johnson, Grand Rrapids, Mich.; A. C. Rogers, Faribault, Minn., and F. P. Gengenbach, Denver.

Friday, June 20—Morning

A joint meeting was held with the American Association of Medical Milk Commissions.

Meeting called to order by Dr. Ogden M. Edwards, Pittsburgh, Pa., President Association of Medical Milk Commission.

Dr. Otto Geier, Cincinnati, read a paper entitled "Report of the Milk Commission on the Relation of Bovine and Human Tuberculosis." No discussion.

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Dr. T. C. McCleave, Berkeley, Cal., read a paper entitled "Certified Milk." Discussed by Drs. J. W. Van Derslice, Chicago; H. L. Coit, Newark, N. J.; John Zahorsky, St. Louis; C. H. Johnston, Grand Rapids, Mich.; T. C. McCleave, Berkeley, Cal.; Ogden M. Edwards, Pittsburgh, Pa.; Otto Geier, Cincinnati, and J. W. Van Derslice, Chicago; Messrs. Stephen Francisco, Newark, N. J., and W. E. Miller, Cincinnati.

Dr. Henry L. Coit, Newark, N. J., read a paper entitled, "The Efficiency of the Milk Commission, Graphically Illustrated." No discussion.

Dr. H. H. Harding, Urbana, Ill., read a paper entitled "The Effect of Barn Operations on the Germ Content of Milk." Discussed by Drs. C. H. Johnston, Grand Rapids, Mich.; T. C. McCleave, Berkeley, Cal.; John Zahorsky, St. Louis; J. W. Van Derslice, Chicago; Otto Geier, Cincinnati; H. A. Harding, Urbana, Ill.; H. L. Coit, Newark, N. J., and Professor Dowling, President Louisiana State Board of Health.

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RADIOGRAPHIC STUDIES OF THE GASTRO-INTESTINAL TRACT IN INFANTS *

HENRY DWIGHT CHAPIN, M.D.
NEW YORK

The difference between a dissection of the body in studying various viscera and organs and the modern Roentgen examination of the same parts will often show quite a field of divergence. The great advantage of the latter method is, of course, that it is dealing with the living and functioning body. Not only are the positions and relations of the organs liable to be somewhat different in life and death, but the mobility and motility of the viscera can be studied by the new method.

This has a practical side, as we are learning that some conclusions drawn from pure anatomy cannot hold entirely good in treating the living body. This is especially true at the beginning of life, when function and form may show wide variations. It will doubtless be necessary for many studies to be made by different observers, with a subsequent comparing of results, before generalizations can safely be made in reference to the limits of location, mobility and motility of the various viscera studied by means of the Roentgen ray. It opens up a promising field of research, however, and we have learned enough already to change our beliefs somewhat as to the form and functioning of the different parts of the gastro-intestinal tract.

In the present study, two cases were observed in reference to the rapidity with which a suspension of barium sulphate could travel from the stomach to the cecum

* Chairman's address.

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and thence through the large intestine to the outlet of the body.

CASE 1.—James L., aged 8 months, weight 14 pounds, 6 ounces. Physical examination negative. Admitted to the hospital for observation as he had had three convulsions several days before. The evening preceding the experiment he was given a dose of castor oil to clear the bowel, and feedings of dextrinized barley gruel started, which were continued through the experiment. The object of stopping milk and giving a fairly soluble food was to avoid possible hard curdling or any digestive disturbance that might interfere with the progress of the barium. At 9:30 a. m. he was given a meal consisting of a heaping teaspoonful of barium sulphate in 8 ounces of acacia by the stomach-tube. An ordinary Roentgen-ray tube was used with an intensifying screen and length of exposure one-tenth second. The infant was placed on the face and abdomen, with arms and legs extended, and Roentgen tube 14 inches from the plate.

A comparison of all the figures shows that up to Figure 5 (three hours and 10 minutes) there is very little gas in the colon. After this time there is considerable gas in the ascending colon and hepatic flexure as well as in the splenic flexure and descending colon. Seven hours after the barium meal the gas begins to disappear from the whole colon, apparently simultaneously with the evacuation of the barium column from the colon into the rectum.

CASE 2.—William J., 7 months, weight 14 pounds, 4 ounces. Physical examination negative. Sent to hospital for loose bowels. This condition was relieved and when the pictures were taken the bowels were constipated with stools rather hard and infrequent. A dose of castor oil was given in the evening followed by dextrinized barley gruel feedings as before. At 9:30 a. m. a heaping teaspoonful of barium sulphate in 8 ounces of mucilage of acacia was given by the stomach-tube.

These two experiments were made on infants of about the same age, weight and general development. In seven hours the barium meal had traveled through the gastro-intestinal tract, as most of the barium column was then evacuated from the colon into the rectum. The end of the barium column is, of course, the measure of

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the motility of the bowel. It must be remembered, also, in these and similar experiments, that foreign bodies, like bismuth and barium, are undoubtedly expelled from the stomach and pass along the intestines more rapidly than normal food. The use of the carbohydrate base tends to prevent interference with stomach acidity.

An interesting account is given by Holzknecht[1] of studies made by him on the motions of the bowel by means of a fluorescent screen. He found that the normal progress of the contents of the duodenum and small intestine is by intermittent, irregular and rapid little jets or jerks. "A cylinder of chyme will be formed, the size of a small coin in section and a finger in length. This will be driven forward for a few seconds at intervals of a minute or two. The suddenness of the movement stretches the intestinal tube, the valvulae conniventes stiffen, and their edges break up and rotate the fluid stream." He further calls attention to the fact that hardly any one has seen the normal peristalsis of the colon. Such peristalsis takes place suddenly, lasting only for a few seconds and occurs at long intervals. He finds, with Stierlin, that these jerks occur from six to eight times in twenty-four hours, the number of displacements varying with different individuals. One case was mentioned in which the whole transverse colon was emptied into the descending colon in three seconds as seen by the fluoroscope.

A third series of cases was studied after a clysma of barium had been given. The location and form of the sigmoid flexure was noted with special reference to the possible passage of tubes. Attention was also given to the distensibility of the colon, the form and situation of the splenic and hepatic flexures, and the patency of the ileocecal valve. Ten infants were thus studied, with thirteen pairs of plates, so that stereoscopic views could be procured. The ages were as follows: 2 months, 9 weeks, 3 months, 4 months, 7 months, 8½ months, 9

1. Holzknecht, G.: Berl. klin. Wchnschr., 1911, xlviii, No. 4, translated in Arch. Roentg. Ray, July, 1912.

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months, 13 months, 18 months and 25 months. An ordinary Roentgen-ray tube was used, as before, with an intensifying screen and length of exposure one-tenth second. Each infant received half an ounce of castor oil four hours before the clysma and was afterward fed with barley water until after the experiment. Eight drops of paregoric were given half an hour before the injection and the rectum painted with a 2 per cent. cocain solution just before administering the barium clysma. This was found helpful in controlling the bowels. Four heaping teaspoonfuls of barium sulphate to each 8 ounces of acacia were injected and the amount retained varied from about 4 to 16 ounces. The feet were held higher than the head and the tube inserted about 4 inches into the rectum, while the funnel through which the barium mixture passed was held about 18 inches above the child. The buttocks were then held tightly together until after the picture was taken. Little barium was usually retained if these precautions were not observed. The picture was started immediately after the injection, averaging about half a minute, the child held face downward on its abdomen and the tube 14 inches from the plate.

The results shown in Experiments 1 to 13 were noted:

Experiment 1 (Fig. 19).—Colon entirely filled, holding 16 ounces. Sigmoid flexure above the umbilicus and falls to the left.

Experiment 2 (Fig. 20).—Rectum, sigmoid flexure, descending colon, splenic flexure and transverse colon filled. Hepatic flexure, ascending colon and cecum outlined with feces, gas and streaks of barium.

Experiment 2 (Fig. 21).—Picture taken fifteen seconds after Figure 20. A peristaltic wave is seen in the transverse colon. The bowel holds 16 ounces. Sigmoid flexure is well above the crest of the ilium, and has fallen over to the right.

Experiment 3 (Fig. 22).—Colon entirely filled. Holds 8 ounces. Sigmoid rises as high as the transverse colon and lies in the form of a figure 8.

Experiment 4 (Fig. 23).—Colon completely filled, holding 16 ounces. Ileocecal valve patent. Sigmoid rises above the umbilicus and shows a complete twist.

Experiment 5 (Fig. 24).—Colon completely filled and some barium in small intestine. Holds 8 ounces. Sigmoid passes nearly to umbilicus then downward to the left.

Experiment 6 (Fig. 25).—Colon, sigmoid and rectum filled. Splenic flexure and lower third of descending colon partly filled. Transverse colon and upper two-thirds of descending colon show streaks. Sigmoid rises to umbilicus and then downward and to the left. Holds only about 2 ounces.

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Fig. 1.—Fifty-five minutes. Stomach horizontal, as observed by the recent studies of Pisek and LeWald. Greater portion of barium in the stomach; considerable amount in the small intestines.

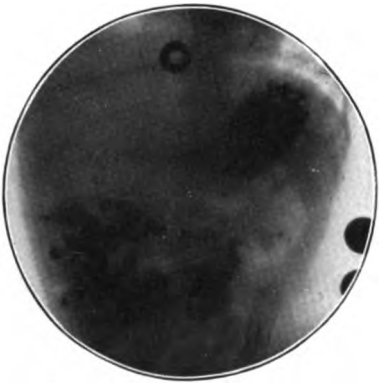

Fig. 2.—One hour and twenty-five minutes. Less barium in the stomach; more in the intestine.

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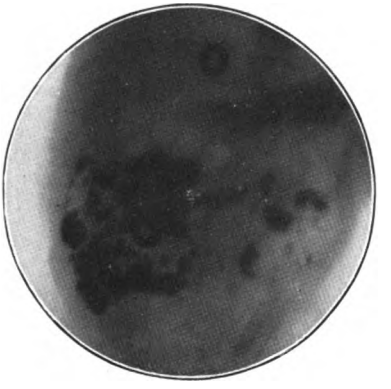

Fig. 3.—One hour and fifty minutes. Small amount of barium in the stomach. The rest of the barium in the small intestines, possibly some in the cecum.

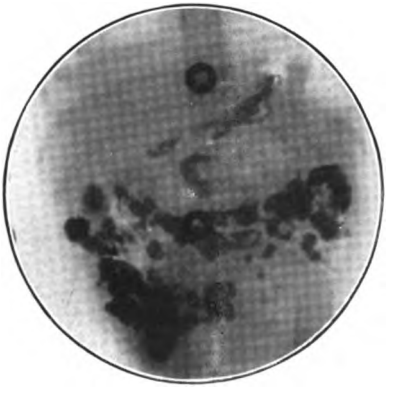

Fig. 4.—Two hours and ten minutes. Very small amount of barium in the stomach; greater part of barium in the small intestines. Cecum fairly well filled.

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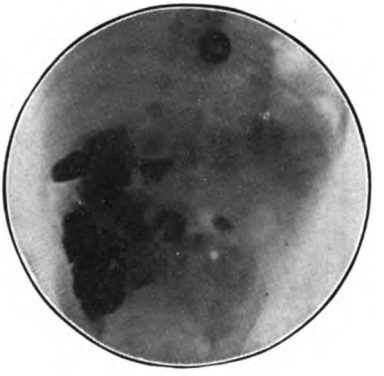

Fig. 5.—Three hours and ten minutes. A trace of barium in the stomach; moderate amount of barium in the small intestine. Cecum, ascending colon, and hepatic flexure almost completely filled, with greater part of barium in these situations.

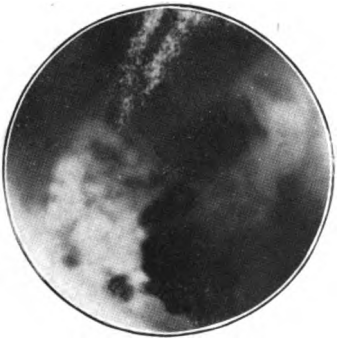

Fig. 6.—Four hours and ten minutes. No barium in the small intestine. Cecum and ascending colon contain the greater portion of the barium. Some barium in the transverse colon which begins to show segmentation.

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Fig. 7.—Five hours. No barium in the small intestine. Cecum and ascending colon well filled with barium. Transverse colon toward the splenic flexure shows segmentation with a small amount of barium.

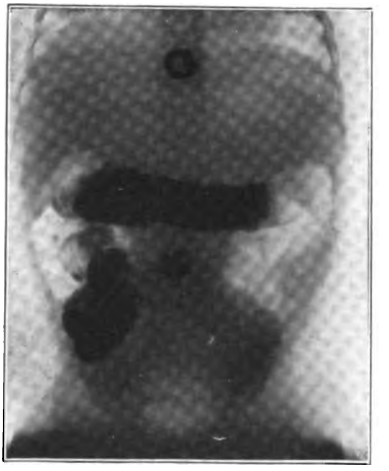

Fig. 8.—Six hours. Cecum, ascending colon and transverse colon to the splenic flexure well distended with barium. Small amount of barium in the sigmoid.

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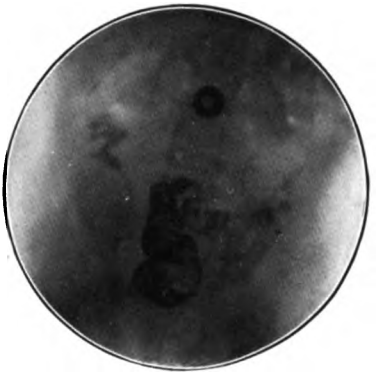

Fig. 9.—Seven hours. Some residue of barium in the cecum. Greater part of the barium has disappeared from the ascending and transverse colon. A considerable amount of barium in the descending colon. Rectum well distended with barium.

Fig. 10.—Twenty minutes. Stomach horizontal. Very remarkable in that a noticeable amount of barium is already in the small intestine.

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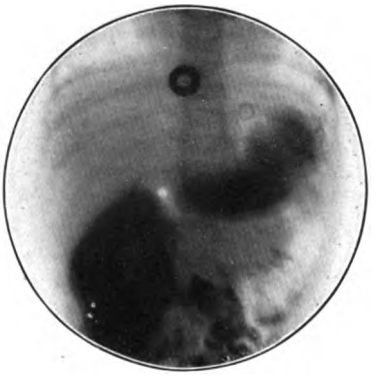

Fig. 11.—One hour and ten minutes. The greater portion of the barium is out of the stomach and is in the small intestine. Stomach has moved higher up and to the left. Barium begins to appear in the cecum.

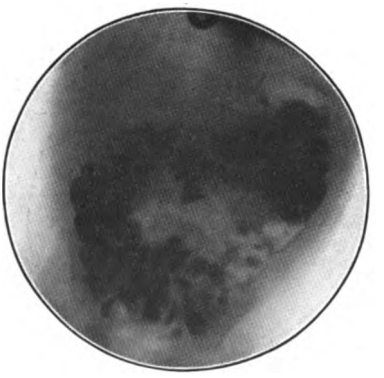

Fig. 12.—One hour and forty minutes. Stomach contains a small amount of barium. Still a considerable amount of barium in the small intestines. Ascending colon, hepatic flexure and a part of the transverse colon contain barium.

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Fig. 13.—Two hours and five minutes. Cecum, hepatic flexure, transverse colon and splenic flexure contain barium. Still a considerable amount of barium in the small intestines and a small amount in the stomach.

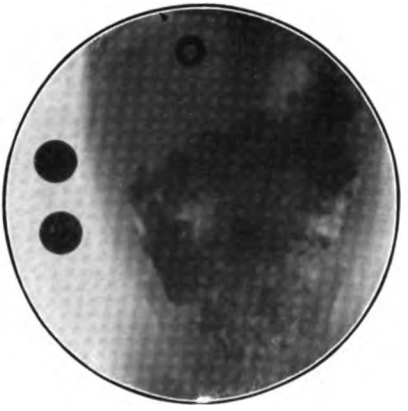

Fig. 14.—Two hours and thirty minutes. Small amount of barium in the stomach. The entire small intestine contains some barium. Cecum well filled with barium and the hepatic flexure, ascending and transverse colon, splenic flexure and first third of the descending colon contain barium.

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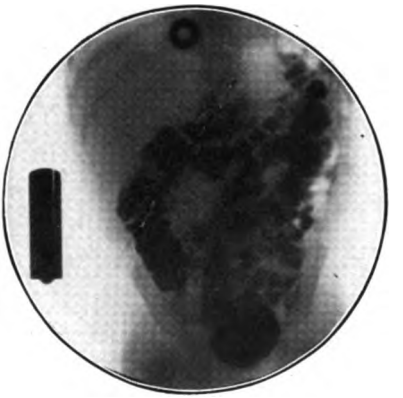

Fig. 15.—Three hours. A small amount of barium still in the stomach. Small intestines contain a considerable amount of barium. The entire colon, including the rectum, contains barium.

Fig. 16.—Four hours. The stomach contains a trace of barium. A small amount of barium in the small intestines. The entire colon mapped out by barium. Noticeable that the cecum and ascending colon contain less volume than in Figure 15. Part of barium has disappeared from the rectum (defecation).

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Fig. 17.—Six hours. A considerable amount of gas in the stomach. Cecum, ascending colon and hepatic flexure well distended with barium. Transverse and descending colon empty. Sigmoid flexure and rectum filled with barium (defecation). This suggests that reverse peristalsis may take place when cecal digestion is not as yet complete.

Fig. 18.—Seven hours Cecum and ascending colon empty. Transverse colon filled near the splenic flexure. Splenic flexure nearly empty. First third of descending colon empty. The rest of the descending colon, sigmoid flexure and rectum filled.

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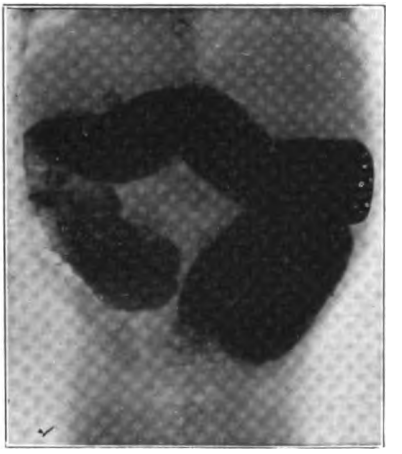

Fig. 19.—For description see Experiment 1.

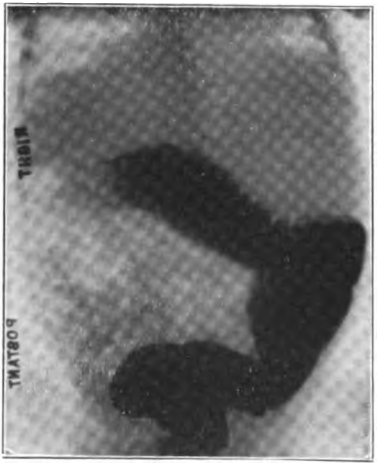

Fig. 20.—For description see Experiment 2.

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Fig 21.—For description see Experiment 2.

Fig. 22.—For description see Experiment 3.

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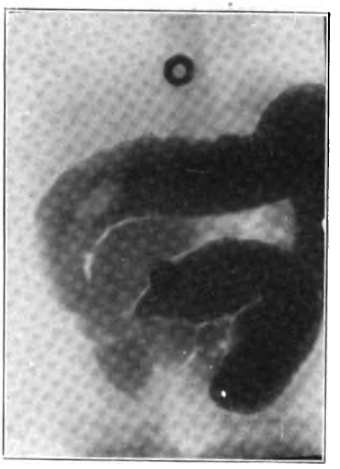

Fig. 23.—For description see Experiment 4.

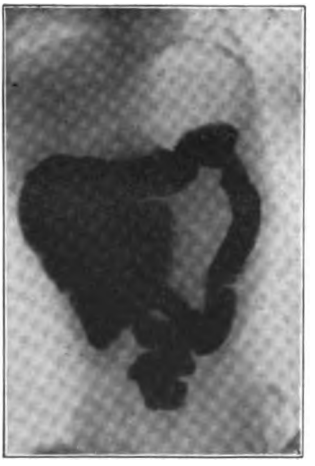

Fig. 24.—For description see Experiment 5.

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Fig. 25.—Congenital dilatation and hypertrophy of colon.

Fig. 26.—Same condition. Transverse colon has sagged into pelvis.

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Experiment 7: Cecum empty. Ascending colon partly filled. Bowel filled from hepatic flexure to rectum. Holds 16 ounces. Sigmoid rises to level of umbilicus and falls to right.

Experiment 8: Splenic flexure, descending colon, sigmoid and rectum filled. Ascending colon, hepatic flexure and transverse colon show streaks. Holds only about 4 ounces. Sigmoid rises to crest of ilium and turns to left of descending colon.

Experiment 9: Lower half of descending colon, sigmoid and rectum filled. Above this, streaks, up to hepatic flexure and the rest empty. Sixteen ounces injected but almost all expelled. Sigmoid rises above umbilicus and falls to right.

Experiment 10: Colon entirely filled from hepatic flexure to rectum. Cecum and ascending colon obscured from sigmoid lying in front. Transverse colon shows peristaltic wave. Sigmoid far to the right, carrying with it the descending colon, apparently due to a long mesocolon. Holds 16 ounces.

Experiment 11: Colon filled, but cecum obscured by sigmoid. Sigmoid rises in front of cecum and above umbilicus almost to transverse colon and then falls to the right. Holds 16 ounces.

Experiment 12: Colon filled but sigmoid obscured. Holds about 8 ounces.

Experiment 13: Colon only partly filled as most of the barium was expelled and picture obscured. In this case the precautions of giving the opiate and cocain to the rectum were not carried out.

It is seen that in these cases there was much difference in various parts of the colon as regards mobility. The hepatic flexure, transverse colon and sigmoid flexure showed the greatest variability. Any injected substances that get past the sigmoid flexure reach the cecum very quickly, and without using much force, as these pictures were taken not more than a minute after the clysmas were given. In one of the cases (Fig. 24) the ileocecal valve was patent, the barium being distinctly seen in the small intestine, and in another case it was doubtful but probable. Dr. James T. Case[2] reports that out of a series of 200 gastro-intestinal cases 33 showed the ileocecal valve incompetent as determined by the barium clysma. Kraus found the ileocecal valve incompetent in only 12 cases out of 150 examinations. He states that the ileocecal valve is patent in new-born infants. This is a subject that warrants future study.

A study of the sigmoid flexure in these cases showed remarkable variations both in form and situation. This structure seems to occupy greater space than has hitherto been supposed. The previous notes show how twisted and doubled-up in all directions it may be during life

2. Case, James T.: Am. Quart. Roentgenol., November, 1912.

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and how it may occasionally reach as high as the transverse colon.

From these and other studies it is evident that it is rarely, if ever, possible to pass a tube through this structure. Nothnagel and Boas, years ago, held that a tube could not be made to pass the sigmoid, and later Lilienthal, in 1906, verified this fact by roentgenograms. The tubes could be passed only to the bend or apex of the sigmoid flexure, when they impinged on the wall, the long mesentery of the sigmoid stretching out like a fan, permitting no further progress. Still later, Soper[3] reported sixty cases in which the position of the tube was verified by radiographs and the tube was always found coiled up in a dilated rectum. In only one case was he able to introduce a tube higher than the dome of the rectum and this proved to be Hirschsprung's disease. The last illustration here shown is an example of this congenital dilatation and hypertrophy of the colon, one of which was later verified at necropsy (Figs. 25 and 26). In Figure 25 the transverse colon has sagged into the pelvis.

SERIES A.

In making these experiments I am much indebted to Dr. Marshall C. Pease for preparing and observing the cases, to Dr. Lewis for taking the pictures in the Roentgen-ray laboratory of the New York Post-Graduate Hospital, and also to Dr. Kast in aiding in an interpretation of the plates.

51 West Fifty-First Street.

3. Soper, Horace W.: The Colon Tube and the High Enema, THE JOURNAL A. M. A., Aug. 7, 1909, p. 426.

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USE OF THE ROENTGEN RAY IN THE DIAGNOSIS OF OBSCURE ABDOMINAL CONDITIONS IN INFANCY AND CHILDHOOD

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BOSTON

I have been much impressed, during the last two years, by the assistance given by the Roentgen ray in the diagnosis of obscure abdominal conditions in infancy and childhood. It not only bids fair to settle definitely the time required for the emptying of the infant's stomach under normal conditions and the effect of the various foodstuffs and salts on this time, but also to be of the greatest value in determining the presence and nature of pathologic abdominal conditions in early life. It has in many instances, in my experience, afforded information which could not have been obtained in any other way, and has made the diagnosis easy in conditions the nature of which could only have been guessed at in the absence of this information. It makes a positive diagnosis easy in many obscure cases, and in many others reveals abnormal conditions which without it would be entirely overlooked.

I shall not attempt to enumerate all the conditions in which the Roentgen ray may be of assistance in diagnosis, but shall merely mention a few in which I have myself found it useful.

It is often extremely difficult to distinguish between chronic gastric indigestion, spasm of the pylorus and

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stenosis of the pylorus in early infancy on the basis of the history and physical examination. The Roentgen ray is of the greatest assistance in differentiating between these conditions and not infrequently gives information in a few hours which could not be obtained in any other way for days or even weeks. It shows conclusively how soon the food begins to leave the stomach, how rapidly it leaves and how long it is before the stomach is empty. The following cases show its value in the diagnosis of these conditions:

CASE 1.—J. M., boy, was born at full term, after a normal labor, and was normal at birth. He was entirely breast-fed and his mother apparently had plenty of milk. He began to vomit when a little over 2 weeks old, the vomiting occurring after every feeding. The amount of the vomitus was at first small, but soon became large. He sometimes vomited while taking the breast, but at other times not until after an hour or more. He at times did not vomit until after the next feeding, when he vomited a large amount. The vomiting eventually became projectile. Changing the intervals between the nursings had made no difference in the vomiting. Barley-water was vomited the same as the breast milk, and during the last four days even water had been vomited. The stools were normal in amount and character during the first three weeks, but since then they had been small, green and slimy. The bowels had not moved during the last three days. He had passed very little urine for two weeks, and had lost weight very rapidly. He was seen when 6 weeks old.

Physical examination showed a fairly developed and nourished baby, of fair color. The fontanel was depressed and the bones of the skull overlapped a little. The abdomen was sunken. The stomach extended 3 cm. below the navel. Marked waves of peristalsis were seen running from left to right after nursing. The baby then had spasms of pain, followed by retching and vomiting. The vomiting was not explosive. No tumor was felt either before or after the vomiting. During the examination he passed a partially formed, medium-sized, greenish-brown stool, which contained considerable mucus, but no definite food elements.

The history in this instance was characteristic of stenosis of the pylorus. The absence of a palpable tumor and of explosive vomiting pointed, however, to spasm of the pylorus. Chronic gastric indigestion was, of course, out of the question. A roentgenogram (Fig. 1) taken immediately after a bismuth meal showed no food leaving the stomach. Repeated pictures, taken at first every fifteen minutes and then every half-hour until six hours (Fig. 2) had elapsed, showed that

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nothing whatever left the stomach during this time. Many of them, moreover, showed definite indentations of peristalsis. He was allowed to nurse ten minutes before the last picture was taken, because feeding will sometimes stimulate peristalsis and cause the stomach to empty itself. These pictures at once established the positive diagnosis of stenosis of the pylorus.

CASE 2.—W. B., boy, weighed 8 pounds at birth. He was nursed for ten weeks, when he was weaned on account of persistent vomiting. A Mellin's Food mixture, equal parts of fat-free milk and barley-water, to which lactose was added, a mixture of fat-free milk and boiled water, to which dextrimaltose was added, and whey were also vomited. He had recently been given eight feedings of 2 or 3 ounces. He vomited immediately after every feeding, although he did not always vomit all that he took. The food was vomited practically as it was taken. He had never vomited a larger quantity at one time than the amount taken at the last feeding. He usually did not vomit more than once after each feeding, but at times vomited several times between feedings. The vomiting had never been explosive. It was, however, often associated with pain. The bowels were constipated, but with the help of milk of magnesia and suppositories he had had one fair-sized, yellow, well-digested movement daily. He had regained his birth-weight in two weeks, but since then had gradually dropped to 6¾ pounds. He was seen when 3½ months old.

He was well developed, but emaciated. The fontanel was depressed. After feeding, the stomach was clearly visible in the epigastrium. The lower border was 1 cm. above the navel, the level above that of the thorax. There was questionable peristalsis, but no tumor was made out when the stomach was either full or empty.

A roentgenogram (Fig. 3), taken immediately after a bismuth meal, showed no bismuth leaving the stomach. One taken forty minutes later (Fig. 4) showed, however, that a considerable portion of the meal had left the stomach. It also showed marked indentations of peristalsis. One, taken at the end of two hours, showed that the stomach was empty. These pictures proved conclusively, therefore, that there was no organic stenosis of the pylorus and that the symptoms were due either to spasm of the pylorus or to a disturbance of the digestion. The development of the vomiting on breast-milk and the persistence of the vomiting on a reasonable diet, together with the visible persistalsis and the marked indentations of the stomach wall shown in the roentgenograms, proved that the case was one of spasm rather than of indigestion.

CASE 3.—M. C., girl, weighed 7 pounds at birth. She had been nursed from the beginning. She was given the breast

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for fifteen minutes every two and one-half hours by day and twice during the night. She had vomited after every feeding since she was a week old. The vomiting sometimes occurred immediately, sometimes about an hour after feeding. The vomiting had not been explosive and she had never vomited more than she had taken at the last feeding. She was apparently always hungry and never satisfied. She had had one smooth, yellow stool daily. Her weight when she was seen, when 2 months old, was a little less than 8 pounds.

She was fairly developed and nourished, but rather pale. The fontanel was level. The abdomen was soft, there was no visible gastric peristalsis and no tumor was felt in the region of the pylorus.

A roentgenogram (Fig. 5) taken immediately after a bismuth meal showed that the food was already beginning to leave the stomach, while one (Fig. 6) taken fifteen minutes later showed that a considerable amount had already passed through the pylorus. The symptoms in this instance, while pointing more strongly to gastric indigestion than to any more serious condition, were suggestive enough of pyloric spasm or stenosis to cause considerable anxiety. The roentgenogram excluded the more serious conditions within fifteen minutes.

There is probably no class of cases in childhood in which the diagnosis is more difficult than that in which there are recurrent attacks of vomiting and abdominal pain, either singly or in combination. In this class of cases also the Roentgen ray furnishes evidence of the greatest importance and in many instances discloses the cause of the symptoms. It not infrequently reveals conditions which could not be determined in any other way. The following cases are illustrative of this class.

Case 4.—A. C., boy, aged 6½ years, had had recurrent attacks of vomiting ever since he was weaned. They had come at first once in six or eight months, but recently every few weeks. The attacks began suddenly with pain in the region of the navel. The pain was more or less continuous. Vomiting always began in less than an hour and persisted. The attacks were always associated with constipation and were relieved when the bowels were thoroughly emptied. They lasted from thirty-six hours to ten days. He was sometimes so weak toward the end of an attack that he did not recognize his parents. He was circumcised in April, 1912, because it was thought that reflex irritation from phimosis might be the cause of the attacks. A diagnosis of recurrent vomiting from acid intoxication was made in one of our

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hospitals in October, 1912, because of the presence of acetone in the urine and breath. His appendix was removed in another hospital in December, 1912, during one of these attacks. They, nevertheless, persisted. He was seen in February, 1913.

He was well developed and fairly nourished. The level of the abdomen was a little below that of the thorax. It was soft and tympanitic. There were no evidences of fluid, no masses were felt and there was no spasm. There was a little tenderness on deep pressure in the left upper quadrant. Rectal examination showed nothing abnormal.

A roentgenogram (Fig. 7) taken while he was lying down, immediately after a bismuth meal, showed that the lower border of the stomach was on a level with the crests of the ilia. Another (Fig. 8), taken six hours later, showed the lower border of the stomach still lower, with considerable bismuth still in it. One (Fig. 9), taken twenty-four hours after the meal, showed the stomach free from bismuth and the colon filled with it. The upper margin of the transverse colon was at the level of the navel. Still another (Fig. 10), taken after a bismuth enema, showed a marked prolapse and consequent kinking of the large intestine.

The attacks were evidently due, therefore (Figs. 7, 8, 9 and 10), to temporary intestinal obstruction, resulting from splanchnoptosis. This condition could not have been recognized in any other way. If the Roentgen ray had been used earlier he would have been spared two unnecessary operations.

CASE 5.—I. R., girl, aged 10½ years, had always been constipated, but had otherwise been very well. She had had repeated attacks of very severe pain in the lower abdomen since the middle of December, 1912. The pain lasted from ten to fifteen minutes and was so severe that she cried out, pulled her hair and lay down and writhed about. The pain stopped suddenly and she then had a strong desire to urinate. She sometimes had four or five attacks in a day, but at other times went as long as a week without one. The attacks were followed by dull pain in the lower abdomen for about twenty-four hours. She had been more constipated since these attacks began, sometimes, in spite of cathartics, going a week without a movement. She was seen early in March, 1913.

She was well-developed and fairly nourished. Her tongue was clean. The level of the abdomen was that of the thorax. It was soft, no masses were felt and there were no evidences of fluid. There was slight tenderness on deep palpation in the median line below the navel. There was no tenderness in the region of the kidneys, and they were not palpable. The

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rectal examination showed nothing abnormal. The urine was of normal color, acid in reaction, and contained no albumin. The sediment showed nothing abnormal.

The symptoms suggested a stone in the bladder more than anything else, although it was evident that the pain might be due to interference with intestinal peristalsis from any one of many causes. Roentgenograms, taken one-half hour (Fig. 11) and two and three-quarter hours (Fig. 12) after a bismuth meal, showed that the stomach was in the normal position and that it emptied itself with normal rapidity. They showed no evidences of stone in either the kidneys or bladder. Another (Fig. 14), taken six hours after a bismuth meal, showed all the bismuth in the cecum and lower third of the ascending colon, proving that there was no interference with the passage of food through the small intestine. One (Fig. 14), taken twenty-four hours after a bismuth meal, showed the cecum and ascending colon empty, while the transverse and descending colon were full of bismuth. The hepatic flexure was only a short distance above the crest of the ilium and the transverse colon sagged down in a V-shape, so that the lowest portion reached nearly to the pubes. The transverse colon was also considerably distended. A roentgenogram taken after a bismuth enema showed the same condition.

The attacks of pain were evidently due therefore (Figs. 11, 12, 13 and 14), to the interference with the intestinal peristalsis resulting from the malposition of the colon, not to a stone in the bladder, peritonitis or pressure on the bowel from the outside.

Case 6.—H. E., girl, aged 9½ years, began two years before, to have acute attacks of pain in the abdomen. These attacks occurred about once in two weeks and lasted about an hour. Vomiting gave immediate relief. There was no apparent relation between the ingestion of food, urination or defecation and these attacks. She continued to have them at irregular intervals for four months. During the next summer she had no attacks, but during the following winter they were more severe and occurred at more frequent intervals. She had no attacks during the following summer and was well until the middle of February, when the attacks again recurred. They were more severe than before and the pain was more definitely located in the left half of the abdomen. During the next month she had two or three attacks a week, the attacks lasting from one-half hour to five hours. Emesis from mustard-water immediately relieved them, but nothing else did. The bowels often moved and she often passed urine during the attack without any relief. She was never jaundiced after the attacks and there was no increased frequency

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of micturition or trouble in passing urine during the attacks. They were not associated with fever. She was seen, in an attack, the middle of March. She lay on her back, writhing with pain, with her legs drawn up and her face pinched. The left upper quadrant of the abdomen was held very tense and there was tenderness in this region. She was immediately relieved by emesis after the ingestion of mustard-water. The tenderness and spasm in the left upper quadrant of the abdomen at once disappeared.

The patient looked perfectly normal in every way. The level of the abdomen was that of the thorax. There was no muscular spasm, no masses were felt and there was no tenderness. The kidneys were not palpable. The liver and spleen could not be felt. The knee-jerks were present and equal. The pupils reacted to both light and accommodation.

The location of the pain, the instant relief after vomiting, and the absence of urinary symptoms pointed strongly to the gastro-enteric tract as the cause of the symptoms. A series of roentgenograms showed, however, that the stomach and intestines were in the normal position and performed their mechanical functions normally. They also showed, however, a stone in the left kidney (Fig. 15), a condition which certainly would not have been expected from the symptoms.

The following case is another example of pain and vomiting in childhood in which a positive diagnosis would have been impossible without the assistance of the Roentgen ray, except with an exploratory laparotomy.

Case 7.—O. L., girl, aged 15½ years, was not nursed when a baby and had much disturbance of the digestion during the first three years. She had always been constipated and for many years had had frequent attacks of colic and abdominal distress. As the result of these attacks, she had been afraid to eat and her diet had become so limited that she was a semi-invalid. When 13½ years old, and again when 15, she had had very severe attacks of abdominal pain, associated with distention and vomiting, lasting for several days. A month before she was seen she came into the hands of a new physician, who found resistance and slight tenderness in the region of the appendix. These symptoms disappeared when her bowels were thoroughly moved. Three weeks later, after a light dinner, she had an attack of pain and distress in the abdomen and vomited about 2 quarts of brownish material, without marked gastric or fecal odor. She was seen a week later.

The patient was short and thin, but of fair color. Her tongue was somewhat coated. The abdomen was sunken and showed nothing whatever abnormal. She stood in normal position, not in that usually assumed when there is prolapse of the abdominal organs.

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Roentgenograms, taken after a bismuth meal, showed on two occasions exactly the same points. There was marked prolapse of the stomach and small intestines, with delay in emptying the stomach. The sigmoid and the splenic flexure were in the normal position. There was marked prolapse of the cecum into the pelvis, with prolapse of the ascending colon. The ascending colon was bent sharply downward on itself for 4 or 5 inches, when it turned sharply upward into the transverse colon, which ran up to the splenic flexure in the normal position. The two parts of the ascending colon were always in the same relation to each other in all positions and at all times. The plate (Fig. 16), taken nine hours after the bismuth meal, showed the stomach empty, but a residue of bismuth in the first part of the duodenum. It also showed the cecum deep down in the pelvis. Those taken twenty-four and thirty-six hours after the meal (Figs. 17 and 18) showed the bend in the ascending colon and also showed that the position of the two parts of the ascending colon to each other is always the same.

These pictures (Figs. 16, 17 and 18) justify the diagnosis of adhesions binding down the colon and involving the duodenum, with presumably secondary prolapse of the stomach and small intestines. The constipation was probably due to the obstruction caused by the adhesions, and the attacks of vomiting to blocking. A laparotomy, two months later, revealed peritoneal adhesions in the places shown by the Roentgen ray.

Constipation is one of the most troublesome disorders of childhood and it is often difficult to determine the cause of the constipation. The Roentgen ray is of great assistance in many of these cases in determining whether or not there is any delay in the passage of the food through the gastro-enteric tract and, if so, where the delay takes place. The following instance shows how much information may be gained from the use of the Roentgen ray in these cases:

CASE 8.—B. J., boy, aged 4 years, was very hard to feed as an infant, a wet-nurse finally having been necessary. He did very well, however, until he was weaned, since when he had had repeated attacks of indigestion, with vomiting. These attacks were always preceded by an increase in the constipation, which was always present. In spite of careful feeding, he required laxatives almost constantly.

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He was in fair general condition and his color was good. His tongue was clean. The abdomen was sunken and nothing abnormal was detected in it. A roentgenogram (Fig. 19), taken immediately after a bismuth meal, showed the stomach in normal position. Another (Fig. 20), taken two hours later, showed that no bismuth had left the stomach during this time. One (Fig. 21), taken six hours after the meal, showed that, while there was a residue still present in the stomach, the greater part of that which had escaped had passed into the large intestine. One (Fig. 22), taken twenty-four hours after the meal, showed the stomach and small intestines empty and almost the whole of the meal in the cecum and ascending colon, very little of it having passed farther on. The constipation was evidently due, therefore, not to sluggish peristalsis in general, but to delay in the colon, the delay in emptying the stomach probably playing no part in the etiology of the constipation.

It is often important to determine whether the colon is in front of or behind an abdominal tumor. Dilatation of the colon with air or water is of assistance in determining this point in some instances. In many others, however, the results obtained in this way are most unsatisfactory. Here, also, the Roentgen ray is often of great assistance. Roentgenograms of the abdomen after a bismuth enema show accurately the position of the large intestine.

Case 9.—These roentgenograms (Figs. 23 and 24) of the abdomen of a baby, 19 months old, with a large sarcoma of the left kidney, illustrate very well the value of this method of examination. The one taken anteroposteriorly shows the bismuth in the sigmoid and in the transverse colon. It is almost entirely squeezed out of the ascending colon and splenic flexure by the pressure of the tumor. That (Fig. 24) taken laterally shows the colon separated from the spine by the tumor.

The Roentgen ray is sometimes of great assistance in differentiating intussusception from infectious diarrhea, in both of which conditions there is blood and mucus in the stools. In some cases of intussusception the roentgenogram taken after a bismuth enema shows a cup-shaped cap of bismuth about the lower end of the intussuscipiens, while in infectious diarrhea the bismuth is generally disseminated throughout the colon. Unfor-

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tunately, however, the picture is not always so plain in intussusception, and it is impossible to be certain from the roentgenogram whether there is or is not an intussusception.

These examples are, it seems to me, sufficient to show how useful the Roentgen ray is in the diagnosis of obscure abdominal conditions in childhood and to illustrate how varied the conditions are in which it is of service. There are probably also many other conditions, which have not been mentioned in this paper, in which it will prove to be of equal value.

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Fig. 1.—Case 1. Taken immediately after a bismuth meal.

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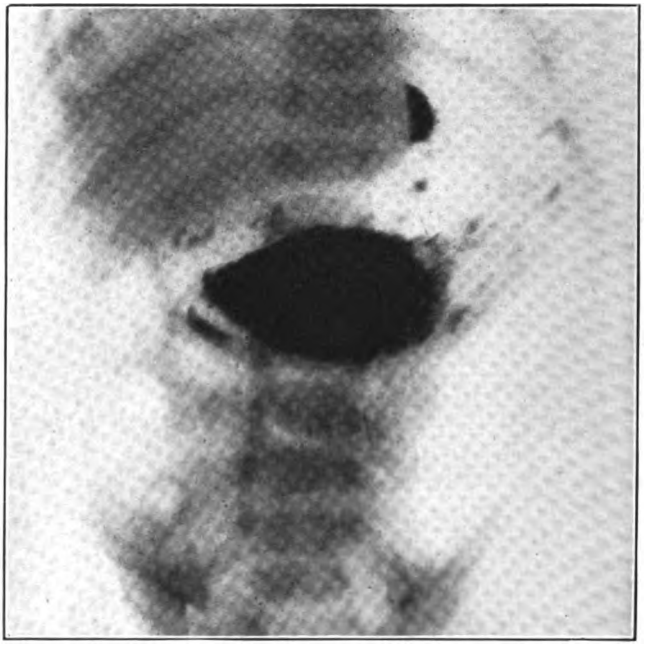

Fig. 2.—Case 1. Taken six hours after a bismuth meal.

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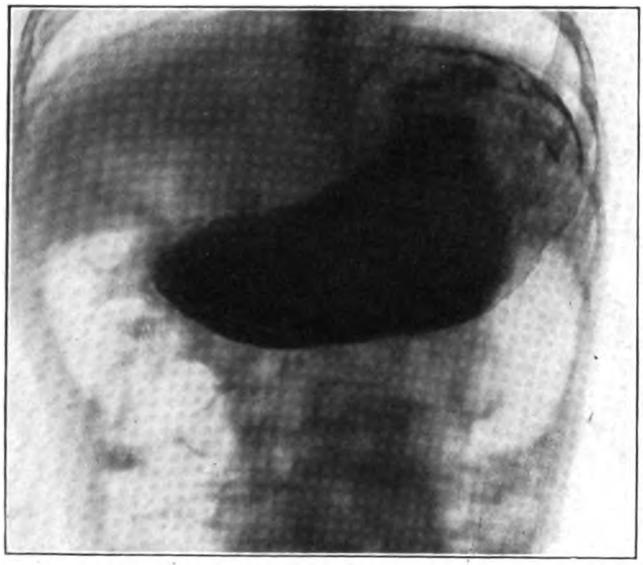

Fig. 3.—Case 2. Taken immediately after a bismuth meal.

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Fig. 4.—Case 2. Taken forty minutes after a bismuth meal.

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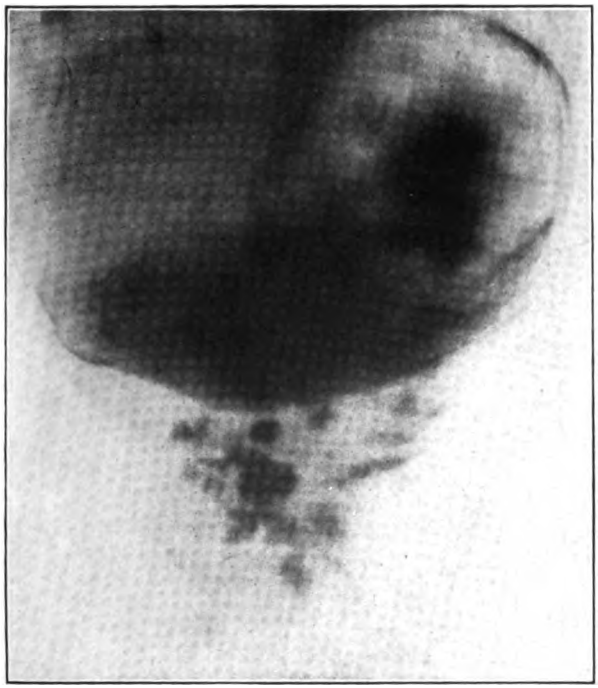

Fig. 5.—Case 3. Taken immediately after a bismuth meal.

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Fig. 6.—Case 3. Taken fifteen minutes after a bismuth meal.

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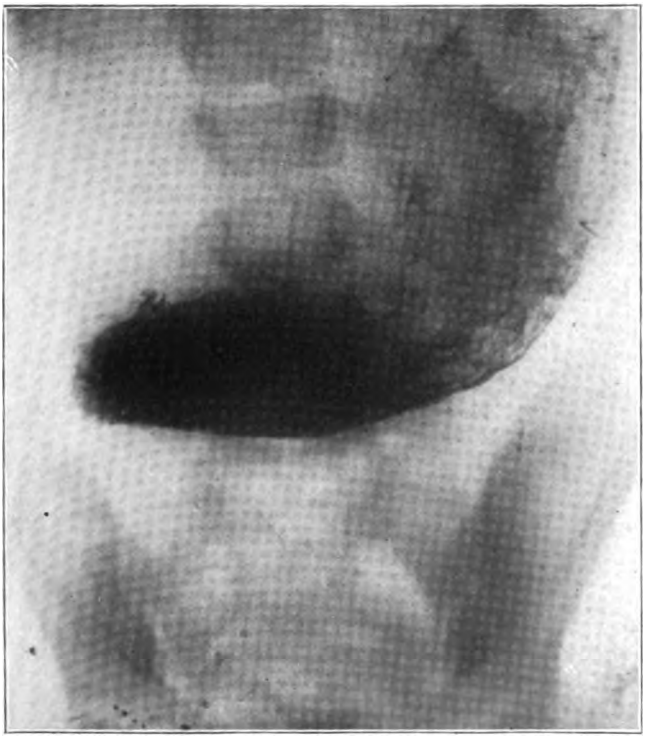

Fig. 7.—Case 4. Taken immediately after a bismuth meal.

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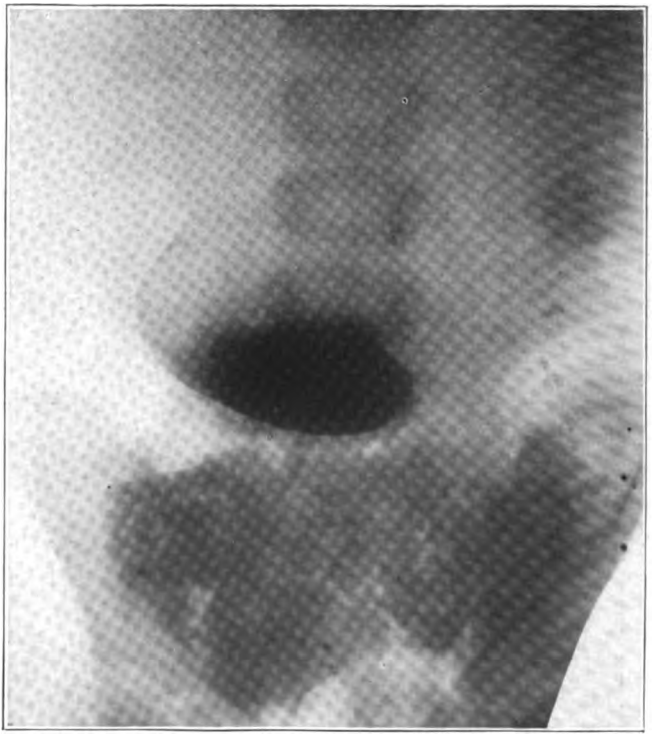

Fig. 8.—Case 4. Taken six hours after a bismuth meal.

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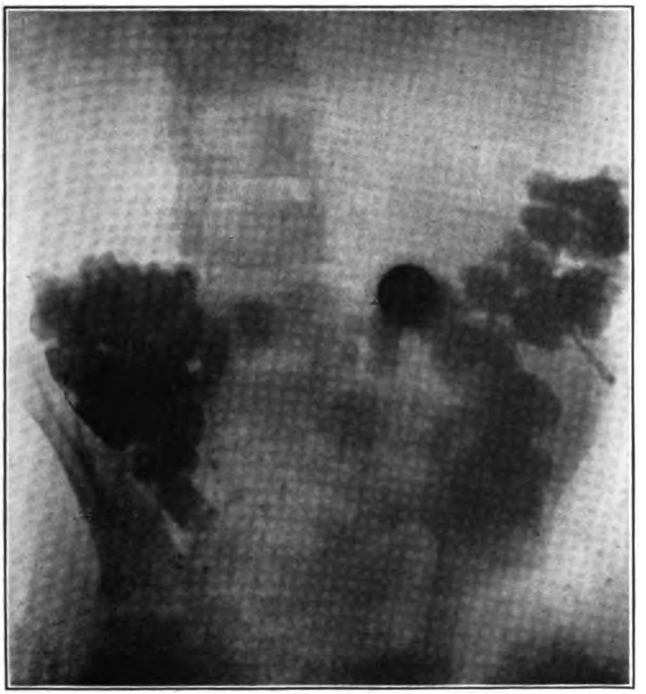

Fig. 9.—Case 4. Taken twenty-four hours after a bismuth meal.

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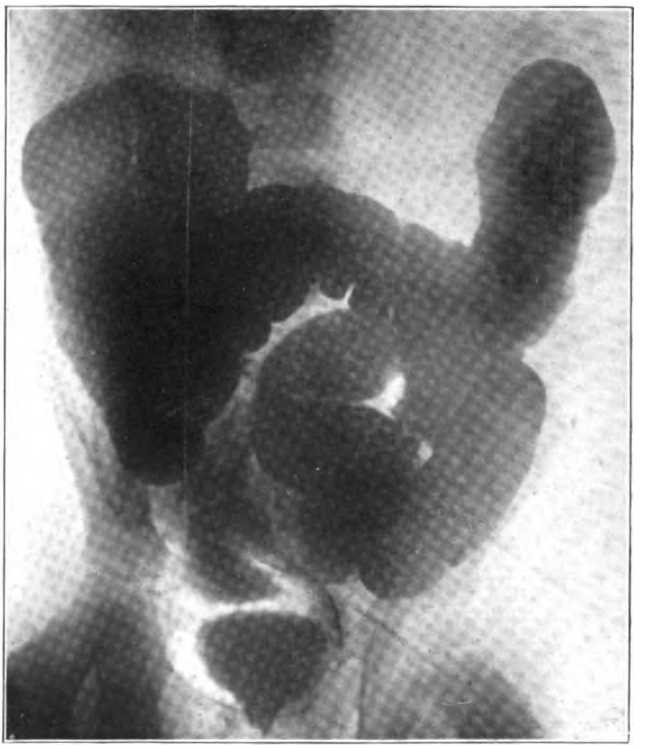

Fig. 10.—Case 4. Taken after a bismuth enema.

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Fig. 11.—Case 5. Taken one-half hour after a bismuth meal.

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Fig. 12.—Case 5. Taken two and three-quarters hours after a bismuth meal.

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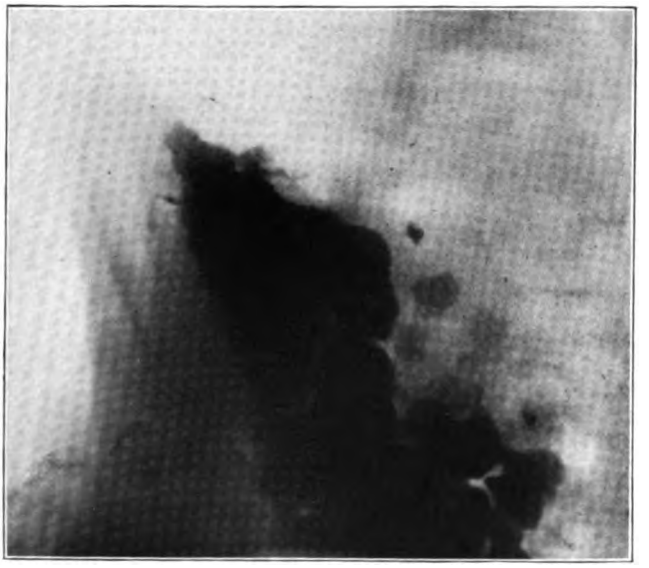

Fig. 13.—Case 5 Taken six hours after a bismuth meal.

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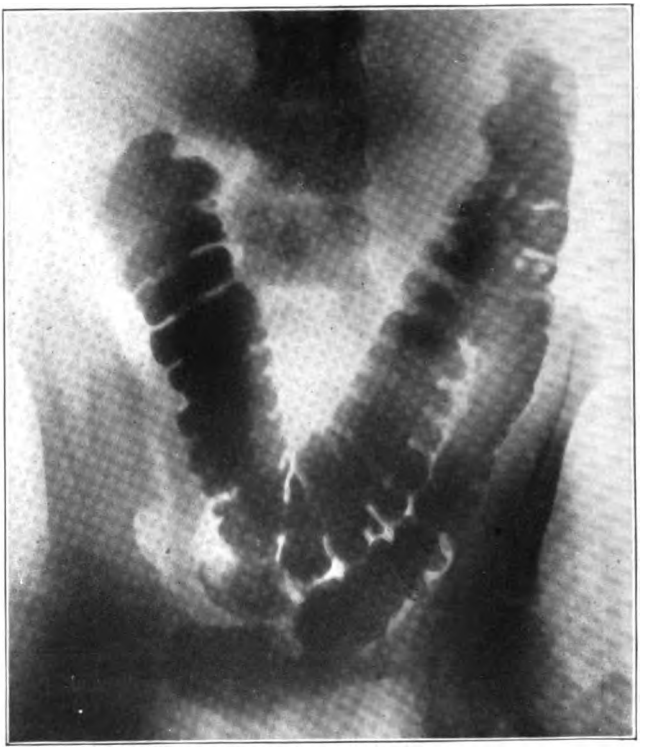

Fig. 14.—Case 5. Taken twenty-four hours after a bismuth meal.

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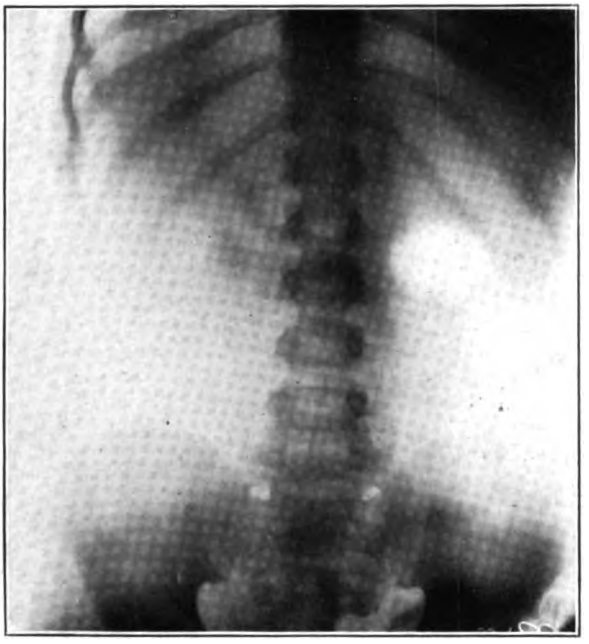

Fig. 15.--Case 6. Stone in left kidney.

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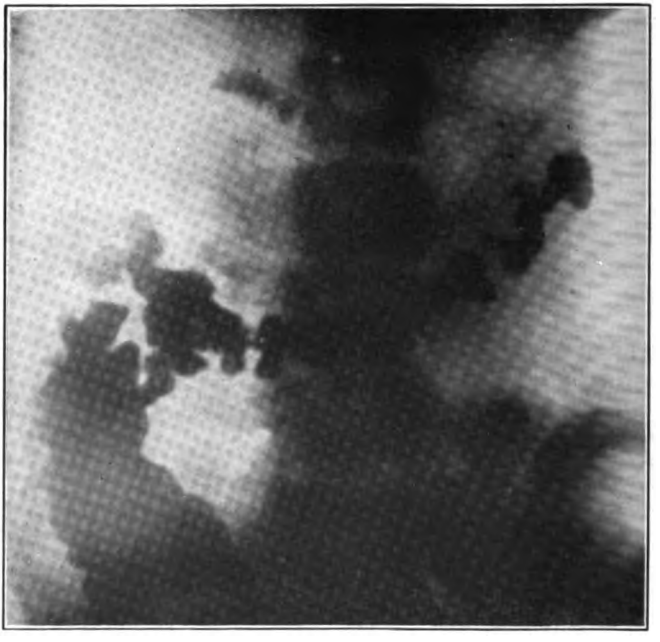

Fig. 16.—Case 7. Taken nine hours after a bismuth meal.

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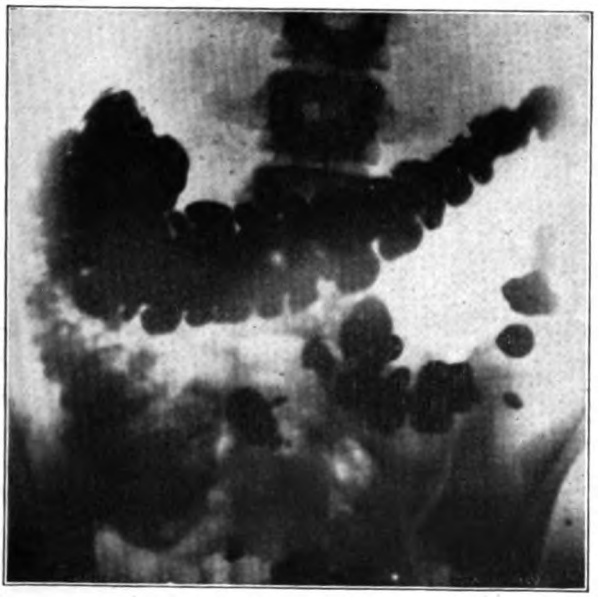

Fig. 17.—Case 7. Taken twenty-four hours after a bismuth meal.

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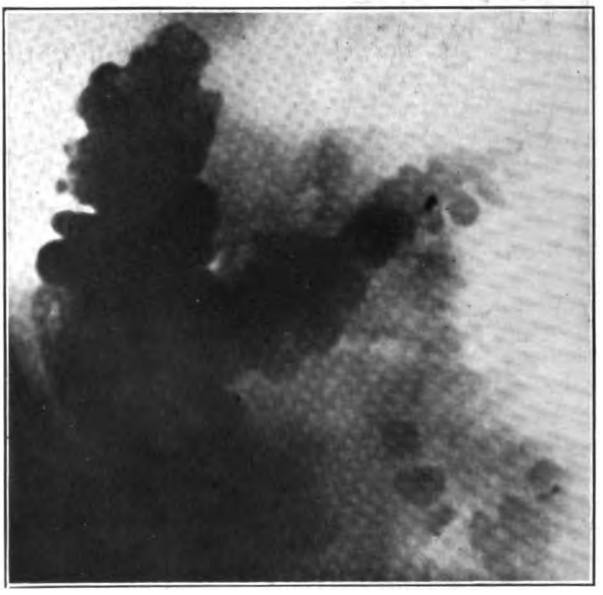

Fig. 18.— Case 7. Taken thirty-six hours after a bismuth meal.

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Fig. 19.—Case 8. Taken immediately after a bismuth meal.

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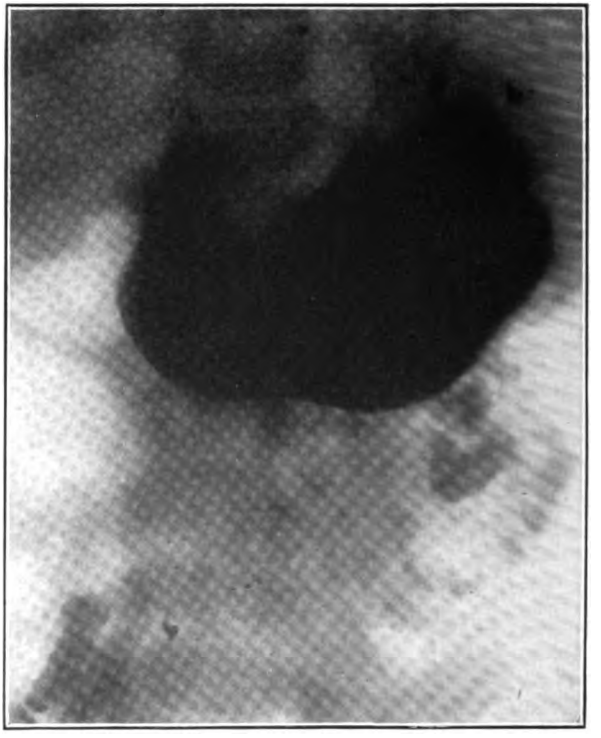

Fig. 20.—Case 8. Taken two hours after a bismuth meal.

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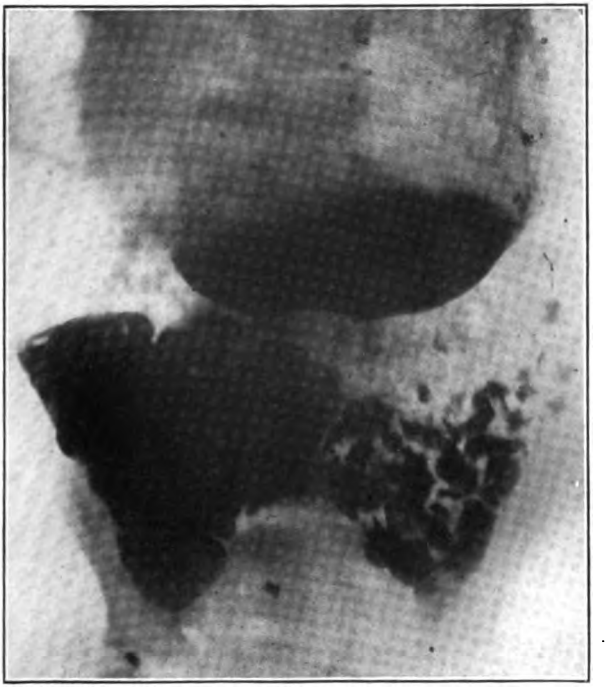

Fig. 21.—Case 8. Taken six hours after a bismuth meal.

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Fig. 22 -Case 8. Taken twenty-four hours after a bismuth meal.

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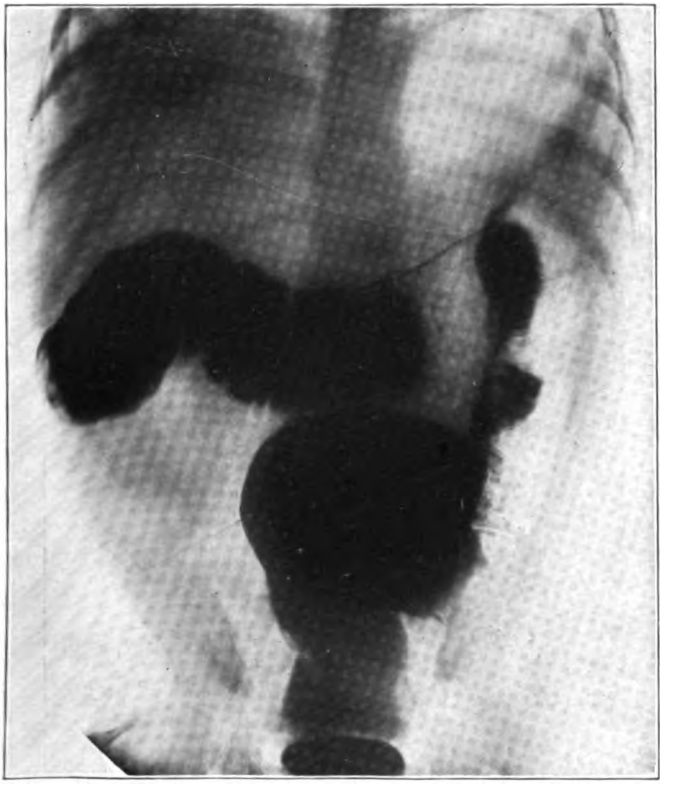

Fig. 23.—Case 9. Taken anteroposteriorly after bismuth enema.

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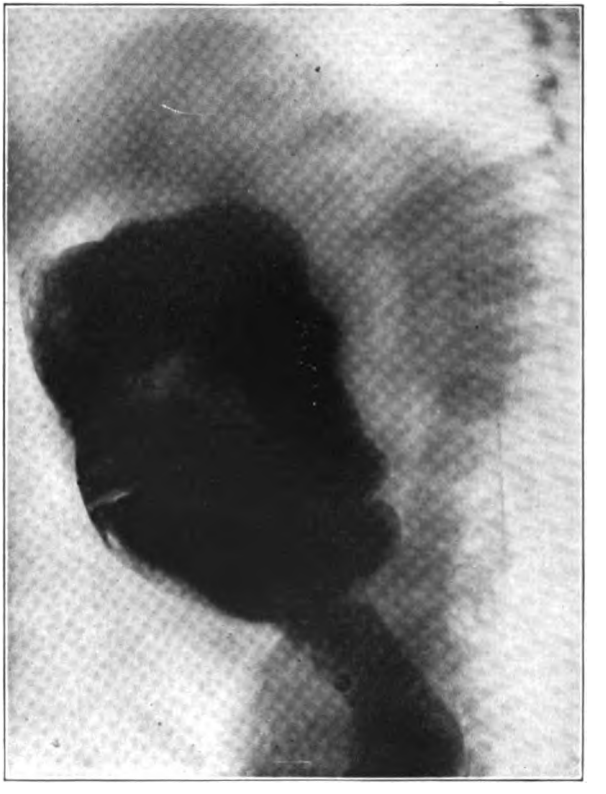

Fig. 24.—Case 9. Taken laterally after bismuth enema.

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THE ROENTGEN RAY IN PYLORIC OBSTRUCTION

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Much information has been gathered since the study of the stomach was begun with the Roentgen ray in conjunction with bismuth. In the adult the size, position, shape, function and presence of tumors may be ascertained with a great amount of accuracy. In the baby, however, the conclusions arrived at in the study of the adult stomach are of limited value. Some work has been done, but much more is yet to be done before the information obtainable by means of the Roentgen ray in the study of the baby's stomach will be had.

The Roentgen ray has revealed to us the erroneous impressions we have entertained, more especially regarding the position, size, shape and function of the baby's stomach. Further studies will show, among other things, the influence of the quantity, quality and composition (including the elements and reaction) of the food given on the function of the baby's stomach. The time the stomach begins to empty itself has been shown to be much earlier than was thought, and the presence of a given feeding has shown that it remains in the stomach longer than we believed. On the other hand, the Roentgen ray with the aid of bismuth corroborates our former clinical observations as to the effect of high fats in retarding the emptying of the stomach. Further study with reference to the causes which influence the various reflexes of the stomach and duodenum will be of interest in illustrating the work already done by the physiological chemist. It will be seen, therefore, that much is yet to be done in the study of the normal stomach before definite conclusions can be determined.

By closer attention to the Roentgen ray and bismuth observations in the examination of the infant's stomach, it is possible that a means of differential diagnosis will be obtained which will be of value, especially

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in young infants, and more particularly in conditions in which obstruction of the pylorus is suspected. It may also explain certain phenomena which make the differential diagnosis between pylorospasm and hypertrophic pyloric stenosis difficult.

While the diagnosis of pyloric obstruction is an easy one to make, it is at times difficult to differentiate between a severe pylorospasm and an hypertrophic pyloric stenosis, and when it is considered that time is a vital element in some instances every means to corroborate a diagnosis is essential. It is believed by some that in a marked case of pylorospasm with symptoms of loss of ground, it should be treated surgically, while others do not concur in this belief. However, if we are reasonably certain that pylorospasm is the condition under consideration, procrastination can better be practiced in the adjustment of food than in stenosis. Before arriving at any definite conclusions in the differential diagnosis by means of the Roentgen ray with the aid of bismuth in these two conditions, it will require a study of many typical cases in which absolute proof is shown either at subsequent operation or necropsy that a tumor is or is not present; and in order that these observations may be of the greatest value they should be made in some uniform way. By this I do not mean that all cases should go to operation in order to secure the information desired, but even in those cases in which the infant progresses satisfactorily much information may be obtained. Not only should the observations be made early, but also later in childhood and in after-life, in order that a better insight into the prognosis of these cases may be had.

There is no doubt that there are two distinct conditions which may cause an obstruction, namely, a pylorospasm and an hypertrophic pyloric stenosis, and that either condition may exist alone. Evidence is plentiful to show also that an hypertrophic pyloric stenosis may have associated with it spasm of the pylorus. The obstruction caused by the hypertrophic pyloric stenosis may vary, as Cautley[1] says, "from a degree which is fatal unless relieved surgically to a slight hyperplasia which is compatible with life."

Various theories and views have been advanced for the etiology of pylorospasm and of hypertrophic pyloric stenosis, but as these do not concern this paper a discussion of them will not be considered. Suffice it to say that pylorospasm is a functional obstruction of the pylorus,

1. Cautley: Brit. Med. Jour., Oct. 13, 1906, p. 939.

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while hypertrophic pyloric stenosis is an organic obstruction due to an hypertrophy of the circular muscular layer of the pylorus, and at times also a slight thickening of the longitudinal muscular layer, with the mucosa swollen and thrown into longitudinal folds.

When the pylorospasm is severe it produces the effect of an hypertrophic pyloric stenosis, while if the hypertrophic pyloric stenosis is mild, but associated with pylorospasm, it may simulate a simple pylorospasm. The inconstancy in the severity of the symptoms in a case of hypertrophic pyloric stenosis is explained in the latter way. In pylorospasm the symptoms cease when the cause is removed, but in hypertrophic pyloric stenosis the obstruction is permanent. Since the hypertrophic pyloric stenosis is a permanent condition, and since many cases are greatly handicapped for life by the obstruction, and since operation does not interfere with the chemism of the gastro-intestinal tract, surgical attention should be promptly given in those cases needing it.

The greatest difficulty lies, as has been said, in differentiating a severe pylorospasm from a mild hypertrophic pyloric stenosis, the reason being the similarity of symptoms. The onset, vomiting, peristalsis, constipation and loss of weight, are found in both. A tumor may be palpated in spasm, and it may not be palpated in hypertrophic pyloric stenosis. Cumston[2] states that pyloric tumor was detected in only 25 per cent. of a long list of cases studied, and Cautley[3] states, "I have seen cases in which the pylorus was said to be distinctly palpable and yet no hypertrophy found after death." Morse[4] states that "it is never safe to conclude that there is no tumor, however, unless the abdomen has been examined with the stomach, both full and empty, and with the abdominal walls relaxed, if necessary, under an anesthetic." It would seem, therefore, that some means other than the palpation of the pylorus is needed to aid in the differential diagnosis of the two conditions causing pyloric obstruction. From observations already made the Roentgen ray promises to fill the need which is so urgent. By its use we will be better able to say that this case should, and this case should not, be surgical, and it is very likely that the mortality, which is now about 50 per cent., will be greatly reduced because the babies will be referred to the surgeon before they become bad surgical risks. It is safe to prophesy that the Roentgen

2. Cumston: Interstate Med. Jour., April, 1911, xviii, 401.
3. Cautley: Brit. Jour. Child. Dis., May, 1908. Reprint.
4. Morse: Am. Jour. Dis. Child., May, 1911, i, 373.

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ray will become a routine measure, as it should be, in the diagnosis of cases of pyloric obstruction.

The ultimate outcome of cases of pyloric stenosis is variable. They must be considered from two standpoints: those operated on and those not operated on. In the first group are those who survive the operation and escape the complications which arise incident thereto, recover, and continue as healthy normal children, though the pylorus, unless a pyloroplasty is performed, remains the same, as has been shown later by autopsy (Morse-Murphy-Wohlbach[5]), and the food leaves the stomach through the stoma as is seen in the cases of Scudder.[6] I know of no instance in which it has been shown that the food leaves the stomach through the pylorus following gastro-enterostomy for complete obstruction. In other words, if the obstruction is so complete as to necessitate operation it remains so. In the second group are: (a) those borderline cases in which the patients have escaped operation, but who would have been much better off had they been operated on; they remain puny and much below par, have occasional gastric upsets, and are inclined to suffer later from gastric dilatation; (b) those patients who have had marked symptoms due to a spasm engrafted on the stenosis and who, when the spasm has subsided, convalesce and become normal children, the stenosis being only of degree; and (c) those children who may have had slight symptoms, but, under the proper care, have convalesced entirely.

With the modern appliances it is not necessary to anesthetize the child for radiographs, as instantaneous pictures can be made; and when it is necessary, an intensifying screen may be used. The observations should be made under as nearly normal conditions as possible. The meal should be at the regular feeding hour and consist of, besides the usual food, a quantity of subcarbonate of bismuth equal to 10 per cent. of the quantity of the meal. If the baby is breast-fed the milk should be withdrawn from the breast, the bismuth mixed with it, and, as in the artificially-fed baby, it should be given by gavage. In this way observations can be made immediately after feeding. If the average capacity of the stomach for the different ages is remembered, and the size of the meal made accordingly, the stomach will not be overdistended and the picture will appear as under normal circumstances. The bismuth does not affect the function of either the stomach or the bowels, and I have

5. Morse-Murphy-Wohlbach: Boston Med. and Surg. Jour., 1908, clviii, 480.
6. Scudder: Surg., Gyn. and Obst., 1910, xxi, 275.

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found that the number of stools per day was neither increased nor decreased.

The position in which the baby is to be placed may be either vertical or horizontal. While the vertical position may give better information because of bringing the viscera covered by the liver more into view, observations made on young infants in this position are made under abnormal conditions. In the prone position the baby may be placed on his abdomen or on his back. Observations in the former position will also be under abnormal conditions, but if the baby is placed in the latter position as he usually lies after feeding, the observations are more apt to be natural. Of course, it is essential that the detail should be brought out so that if it be necessary the position should be changed to obtain the best results, always endeavoring to have the conditions as nearly normal as possible.

The information to be derived from the radiographs regarding the stomach is the position, shape, size, size of flow through the pylorus when the flow begins, and the length of time required to empty the stomach completely. An absence of shadow is also significant.

The stomach occupies the left hypochondriac region and extends through the epigastric region to the right where the pylorus is overlapped by the liver. Its shape is variable as has been shown by Pisek,[7] and may be of the Scotch bag-pipe, tobacco-pouch, pear, or retort shape. The adult fish-hook type is found only after the second year. The position in which the observations are made may influence the shape, as is shown by comparing the radiographs of Pisek[7] with those of Ladd.[8] The stomach contracts in a particular manner in the normal infant stomach: instead of the peristalsis which is seen in the adult, the stomach contracts in all directions in a squeezing manner. Nature seems to provide against any injury to the stomach by having an air space which acts in a pneumatic way. It is possible that its presence may be the explanation of the projectile vomiting of the baby when there is an obstruction at the pylorus. The size of the stomach varies according to age under normal conditions, and it may be seen dilated or contracted under abnormal conditions.

The rapidity with which the food leaves the stomach is surprising: frequently by the time a meal is finished and an exposure made, some

7. Pisek: Trans. Am. Ped. Soc., 1913.
8. Ladd: Trans. Am. Ped. Soc., Washington, May 9, 1913.

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food has already escaped from the stomach. Position of the body does not seem to affect it. It is not always possible to determine the size of the flow through the pylorus, probably because the flow of food is not continuous. The food remains in the duodenum only a short time, passing through spasmodically so that the duodenal stage may also be missed. The length of time required for the stomach to become completely empty has been shown to be variable. Leven and Barret,[9] in a large number of infants from 2 to 6 months old, found the time to vary between one and three-quarter hours to two hours for both woman's and cow's milk. Pisek[7] believes the probable emptying time to be less than three hours, and the average final emptying time to be three hours. There are many factors to be considered, however, which influence the emptying time of the stomach. Ladd[10] states that the stomach appears to empty itself of the greater part of its contents from one and one-half to two and one-half hours and that the residue is emptied very slowly, often after remaining for four, five or even seven and one-half hours in both breast-fed and bottle-fed babies. An interesting point is that the residue from the bismuth meal does not appear to mix with a subsequent meal given three hours afterward. Experiments of Ladd[11] show that high fat, fat-free milk and milk with a high protein content retard the emptying of the stomach.

In obstruction of the pylorus the radiographs show that the shadows are frequently different from those of the normal stomach in size, shape and position, but more particularly the serial radiographs show the greatest differences to be in the function of the stomach. Observations recorded have been very few, but from the study of them the following may be said: In spasm the shape may not be altered, and in stenosis also the stomach may not be altered in shape, but at times it appears as a round ball (Murphy[12]); both conditions may give the characteristic waves of obstruction; in spasm the position may be the same as in the normal stomach, but in stenosis the shadow of the stomach is frequently limited to the left side and there is no shadow in the pylorus or duodenum; if the stenosis is not complete the stomach will appear as in

9. Leven and Baret: Radioscopie Gastrique et Maladies de l'Estomac, 1909, p. 173.

10. Ladd: Am. Joub. Dis. Child., 1913, v, 349.

11. Ladd: Am. Joub. Dis. Child., 1913, v, 357.

12. Murphy: Surgical Clinics of John B. Murphy, 1913, ii, No. 1.

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spasm; the function of the stomach in spasm is changed in a degree, that is, the food may be slower in beginning to leave the stomach and the stomach is emptied spasmodically; it also may require a little longer time for the stomach to empty, whereas in stenosis the food leaves the stomach extremely slowly in some cases and not at all in others, depending on the degree of the stenosis. In Ladd's[13] case a very small quantity of the bismuth was seen in the intestine in two hours, and in Scudder's[14] case no bismuth had left the stomach in four hours.

In only three of four cases coming under my observation during the past ten months have I been able to obtain radiographs. While the number is small I believe that each case is of sufficient interest to report.

CASE REPORTS

CASE 1.—Charles was born on Sept. 9, 1913, sixteen days before he was expected, and was delivered by abdominal cesarean operation. He weighed 7 pounds 10/16 ounce. He was seen by me on the day of his birth. The family history was negative, with the exception of a miscarriage due to a fall. The first day the baby was given water, which he vomited. On the second day he was given 1 ounce of a 6 per cent. sugar of milk solution at two-hour intervals, which he vomited at times. On the third day he was put to the breast. He frequently vomited the breast milk. As the mother had very little milk and the baby was losing weight, a complemental feeding consisting of a 1 per cent. fat, 7 per cent. sugar, .90 per cent. protein and 6 per cent. alkalinity was given. The baby did not seem to care for his food and at times refused to take it. His first yellow stool was passed on the eighth day. His weight at this time was 6 pounds 14 ounces. Not knowing just what he was getting, observations were made to determine how much milk he obtained from his mother. By weighing him before and after nursing it was found that he would get from ¼ to ½ ounce at each feeding. A sufficient quantity of the complemental feeding was given to meet his requirements. This method of feeding was pursud until the sixth month, the quantity and strength of food being increased from time to time, always approximating the capacity of the stomach. The alkalinity was lime water equal to 50 per cent. of the amount of milk and cream. From the time this was begun he commenced gaining and at his sixth month weighed 14 pounds 6 9/10 ounces.

On September 16 typical peristaltic waves were noticed, and while the explosive type of vomiting became more pronounced, no tumor was positively palpated. From this time on the peristaltic waves and explosive vomiting became more and more characteristic. At times he would vomit over the side of the crib to a distance of 3 or 4 feet. On September 21 a crowing sound was first heard when he was nursing. It became significant later as indicating that his stomach was full, as evidenced by explosions if nursing was continued. By careful feeding, particularly as to composition, alkalinity and quantity, it was possible to minimize the number of vomiting attacks, so that he frequently would have only one a

13. Ladd: AM. JOUR. DIS. CHILD., 1913, v, 355.
14. Scudder: Surg., Gyn. and Obst., 1910, xxi, 285.

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day. His weight was noticed carefully and showed a daily gain. His stools were normal, there being only one daily. The explosions continued from time to time, gradually becoming less frequent until January 18, when the last one occurred. The waves became less and less evident as the baby became fatter. A discomfort noticed after feeding when the waves were apparent, however, continued after the waves could no longer be seen.

Jan. 19, 1913, the radiographs herewith presented were made. His usual feeding of 6 ounces of a 1.5 per cent. fat, 7 per cent. carbohydrate, 1.29 per cent. protein was given, to which was added three teaspoonfuls of subcarbonate of bismuth. He was placed in a horizontal position on his back. As will be seen, his stomach is functioning nearly normally. Bismuth was found in his stool five hours after the bismuth was given him, and he had one stool as usual on that day.

At present the baby is apparently healthy and his stomach functions normally. When last seen at the age of 7½ months he was taking 8 ounces of a 3 per cent. fat, 7 per cent. carbohydrate and 2.10 per cent. protein at three-hour intervals. and a cereal jelly once daily. He weighed 16 pounds. On one or two occasions when he had some acute infection of his nasopharynx a mercurial purgation followed by castor oil was given and was well borne.

Case 2.—Amelia was seen in the out-patient department of Touro Infirmary on Dec. 17, 1912, when she was 5 weeks old. She was born at full term and weighed 7 pounds. There was a luetic family history. She was breast-fed, but had frequently vomited since birth. Her weight when first seen was 8 pounds 4¼ ounces. Her stools were black until the third day. She was small, had a wig, the posterior bosses were prominent, and there was scaling of her palms and soles. Otherwise the examination was negative. On December 21 the Wassermann was negative. She had been having three to four green, watery stools daily. She improved slowly, vomited occasionally, and her stools improved in character and decreased in number. She was weighed before and after nursing the breast and showed a gain of only 2½ ounces. At her third month she was constipated. She vomited after, and also between, nursings. March 18 the mother reported that the baby vomited frequently, the vomiting being explosive in character. She was having three to four stools daily, and had lost weight. Her weight was then 9 pounds 8 ounces, having lost ¾ pound since February 24.

Peristaltic waves could be seen, but owing to the rigidity of the abdominal walls palpation was unsatisfactory. No tumor was felt. She got only 2 ounces at each nursing at this time. She was ordered 6 ounces of a weak mixture of a 1 per cent. fat, 7 per cent. carbohydrate, .90 per cent. protein, and 6 per cent. lime water every 2½ hours. March 25 she did not retain the food, had green stools and explosive vomiting continued.

April 5 the Wassermann was positive. The following day the mother was given 6 decigrams of salvarsan and the baby was allowed only breast milk.

April 8 radiographic observations were made. She was given 6 ounces of milk mixture with three teaspoonfuls of bismuth subcarbonate and the accompanying radiographs were taken. She was placed in the horizontal position on her back. Bowels did not move until twenty hours after the observations were made and bismuth was found in the stool.

April 26 no explosions since mother was given salvarsan, and since inunctions of mercury were begun two weeks before. At present the baby is continuing to improve, takes all food and is much better in every way.

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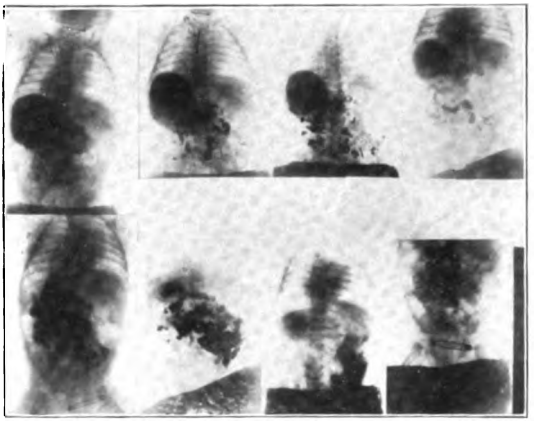
1 2 3 4



5 6 7 8

Fig. 1.—Charles. Taken immediately after giving food by gavage. In this and the following illustrations, the plate was in front, and the tube behind the patient, so that the roentgenograms are viewed postero-anteriorly.

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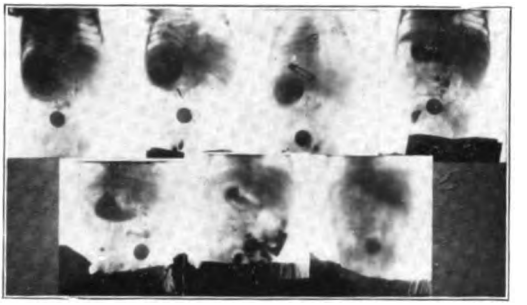
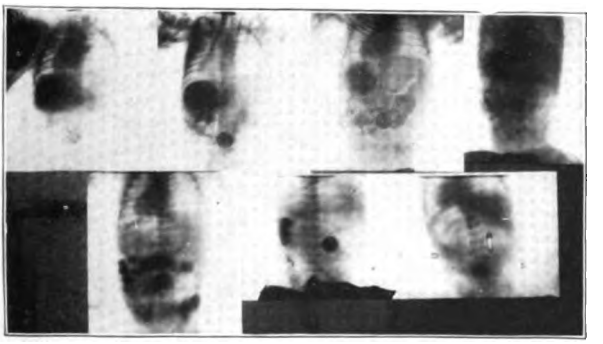


5 6 7

Fig. 2.—Amelia.

1 2 3 4



5 6 7

Fig. 3.—Lillan.

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CASE 3.—Lilian was born Feb. 16, 1913, at term. The confinement was normal and the baby weighed 6 pounds 12 ounces. She was first seen March 4 because she was not doing well. She was breast-fed and vomited at times. She had several green, watery stools daily. She weighed 6 pounds 12 ounces. Family history was negative and physical examination showed a small baby with pinched expression, poorly nourished. Otherwise the examination was negative. She was given a complemental feeding of 2 ounces of 1 per cent. fat, 7 per cent. carbohydrate, .90 per cent. protein and 6 per cent. alkalinity.

The infant was again seen March 11 when I was called on account of the illness of her brother. Because of the baby's appearance the mother was instructed to weigh her before and after nursing in order to determine the quantity of food that the baby was getting, and to continue the complemental feeding, because she had gained only 3 ounces since March 4 and apparently was not thriving. For various reasons the observations were delayed and it was not until March 21 that she was seen again. An examination of her mother's milk showed 6 per cent. fat and specific gravity 1.030. The symptoms became more marked and on March 22 a typical explosion was seen. It was said that she had had such explosions before, the first explosion having occurred shortly after her birth. At this time she weighed 7 pounds 12¾ ounces. Peristaltic waves were seen. The breast-milk was ordered withheld for a few days in order to determine whether it was the cause of the vomiting, and the baby was given the complemental food as already ordered, except that 4 ounces were given instead of 2 ounces, with lime-water equal to 50 per cent. of the amount of milk and cream, every two hours. She began gaining, though very slowly, and on March 31 weighed 8 pounds 1 3/8 ounces. The symptoms continued and became more marked, there were frequent explosions, and peristaltic waves were constantly seen during and after feeding. Her bowels moved six times daily; stools well digested but soft. The mother had allowed her breasts to stop secreting milk. A wet-nurse was considered, but because of the radiographic findings and the gain in the baby's weight artificial food was resorted to exclusively. A satisfactory palpation of a tumor was obtained.

On April 6 she was given 3 1/3 ounces of her regular formula (which was at the time a 1.33 per cent. fat, 7 per cent. carbohydrate, 1.20 per cent. protein, 17 per cent. lime-water), with two and one-half teaspoonfuls subcarbonate of bismuth. The radiographs herewith presented were then made. She was placed in a horizontal position on her back. Bismuth was found in the stools three hours after it was given. She had six stools, as usual, the day of the observations.

At present she is progressing very satisfactorily, having gained 1 pound 8⅛ ounces in the previous twenty-two days. The last explosion occurred on June 1, but waves can be seen during and after each bottle. The cinematograph pictures were made June 12, 1913. She is now taking 5 ounces of a 1.80 per cent. fat, 22.5 per cent. carbohydrate and 1.8 per cent. protein at each feeding, and is having two perfect stools daily.

DISCUSSION

Early in the case of Charles (Case 1) I was inclined toward the belief that he had a congenital hypertrophic pyloric stenosis because of the history, the promptness with which the symptoms began, the constipation, the projectile vomiting and the peristaltic waves. The loss of

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weight during the first three days was not extreme, but I believed that it was due to having begun promptly with the feeding. The inability to palpate the tumor may have been due to the fact that there had not been much wasting with the consequent thinning of the abdominal walls, making palpation more satisfactory. However, realizing the infrequency of palpating the tumor, failure to find it was not considered unusual. The slow gain in weight may have been found in other conditions. At 1 month the baby weighed 8 pounds, or 8¼ ounces more than at birth, which was believed to be satisfactory considering existing conditions. As the baby improved, the symptoms becoming less marked (though they persisted); the possibility of a pylorospasm being the cause of the symptoms was not lost sight of. On January 19, though the baby was progressing particularly well, it was decided to determine the function of the stomach. The evidence obtained led me to believe that the symptoms may have been due in great part to spasm of the pylorus because of the excellent functioning of the stomach; but the continuation of peristaltic waves and the prolonged stay of the bismuth in the stomach (more than five hours) were sufficient to warrant a diagnosis of hypertrophic pyloric stenosis a degree with probable spasm.

In the case of Amelia (Case 2) the feeding, vomiting and stools would suggest a digestive disturbance, but the projectile vomiting and peristaltic waves, with the other symptoms, would indicate a pylorospasm. However, the luetic condition may have played an important rôle in this case. The slowness with which the food left the stomach, the length of time it remained in the stomach, and the waves suggest an obstruction of the pylorus. The fine duodenal stream may also be indicative of some obstruction. The peculiar circle in the region of the pylorus and duodenum may be significant. The radiographs shown in this case, the symptoms, family history, personal history, and the amelioration of the symptoms coincident with the beginning of antiluetic treatment (the baby taking food after, which before the treatment she could not take), strongly point to the luetic condition as being an influencing factor at least in producing the obstruction (probably pylorospasm) which was the cause of the symptoms in this case.

In Lilian's case (Case 3) the condition is one more especially of pylorospasm though there may be a degree of hypertrophic pyloric stenosis. The onset of the symptoms, the characteristic vomiting and peristaltic waves are indicative of some obstruction. The frequent stools

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would suggest a spasm, but the presence of a palpable tumor, while it may be rarely felt in spasm, is more suggestive of an hypertrophic pyloric stenosis. However, with the evidence obtained with the Roentgen ray, the position of the stomach, the early outflow of bismuth and emptying of the stomach, the condition is undoubtedly due in a great measure at least to pylorospasm.

I wish to express my thanks to Dr. E. C. Samuel for the radiographs presented in this paper.

1776 State Street.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. CHAPIN, MORSE AND DE BUYS

DR. ABRAHAM JACOBI, New York: Dr. Chapin referred to some conditions which have been known in former years simply from a physiologic and anatomic point of view. In connection with the two cases which he calls Hirschsprung's disease, he mentions protracted constipation, and, from his illustrations of the lower part of the colon and sigmoid, I concluded that some of these cases were undoubtedly those that forty years ago were called by me congenital constipation. That means a condition of the sigmoid in which it is so long that it forms not one convolution only, but two or more, and absolute constipation is the result. When you find a baby that has been constipated from birth, a constipation lasting for months or years, with a mother who has the full amount of breast milk and who gives the baby no other food, you have reason to conclude that the case is one of congenital multiple sigmoid flexions. When the sigmoid is so long it bends on itself, and sometimes absolute obstruction occurs. This is, however, not to be called Hirschsprung's disease, which means a congenital dilatation of the colon, the genuine megalocolon, which is a rare occurrence.

DR. G. R. PISEK, New York: It is significant that we should have three papers dealing with this method of diagnosis in one afternoon. It proves the increasing value of the method and should emphasize the importance to all of us of the use of the Roentgen ray in pediatric work; and particularly urge us to take advantage of the newer methods that experts in this line are developing. In other words, the fact that we can take instantaneous serial pictures, without any danger whatever to the little patient, is of inestimable value, particularly in conditions dealing with pyloric spasm and pyloric stenosis. Indeed, if this were the only use, it would be worth while, for it enables us to differentiate the two conditions, tells us when to put the case into the hands of the surgeon or satisfies us that it can be treated medically.

I was glad to see Dr. Morse's pictures of gastroptosis. It has occurred to me that possibly such diseases as psychic vomiting may also be found by these methods to be of anatomic origin. Possibly we may find that a number of these related conditions are precursors of diseases of the digestive tract in later life.

DR. L. G. COLE, New York: I should like to ask Dr. Chapin if he considers that the contraction, which he obtained in two cases after giving an enema, is the same thing that Holzknecht described as occurring when food is given by mouth and allowed to progress through the gastro-intestinal tract in the regular way, or whether he considers the contraction he has shown as a spasm from unusual filling of the colon from below.

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Dr. A. W. Crane, Kalamazoo, Mich.: It is gratifying to see Roentgen-ray methods invading the realm of general practice, not excepting pediatrics. In the past children have been bad subjects for roentgenoscopy, but with instantaneous exposures conditions have changed. Roentgenography will, however, fail to impress the general practitioner as a true clinical method of examination. It is the fluorescent screen which gives us true moving-picture images of the stomach and intestines, as well as of heart, lungs and diaphragm, and provides us with a direct clinical method of examining the child.

The first objection that might be suggested to you should be the danger of exposing young tissues to the Roentgen ray. This danger may be minimized by using the smallest amperage which will give a clean-cut shadow, and the slowest number of interruptions which will admit of shadows without flickering. In this way roentgenoscopy extending from five to ten minutes will be comparable in quantity of ray used to that used in instantaneous work, in which several roentgenograms are made. Dr. Cole of New York is one of the best qualified men in the United States to discuss this question of serial roentgenograms. He has recommended that no studies of the stomach be made without ten or twenty roentgenograms of the stomach in series. If we use a screen, plates are not, as a rule, needed.

Only by repeated studies on the part of many pediatricians, can the mass of knowledge necessary to interpret roentgenograms be worked out. The large number of cases that has come before the Holzknecht X-Ray Clinic in Vienna has enabled them to work out a uniform technic whereby a normal or diseased stomach under certain conditions would present certain definite pictures to the examiner. It is only by having a uniform technic that we can classify results and arrive at a collective experience of value in the examination of the stomach and intestines. It seems to me that those who have witnessed screen examinations in a dark room and have palpated the stomach and colon while they are under observation, can realize the transcendent value of this method. There is no other clinical method which offers so much for the future of diagnosis.

Dr. Henry F. Helmholz, Chicago: I should like to ask Dr. Morse about the case with the "V"-shaped transverse colon. I have noticed in post mortems on children that this condition is quite frequent. I wonder whether he has roentgenograms of a large number of cases to indicate how often this occurs.

Dr. H. Lowenburg, Philadelphia: While I agree that it is of great importance to make roentgenographic studies of these cases, it is wrong to give the impression, especially with reference to cases of pyloric obstruction, that they must necessarily be studied in that way in order to determine the diagnosis, prognosis and treatment. I was particularly interested in Dr. Morse's paper. With reference to his first series, I think the same results can be obtained by the administration of charcoal. You can test the patency of the pylorus by the administration of charcoal and whether or not it can be recovered in the feces. This depends on the degree of obstruction. If charcoal is recovered the next day in the stomach washings it indicates considerable or complete obstruction, and the possibility that the case will become surgical is made greater. Absence of charcoal in the washings lessens the possibility of operation. I think that the greatest field for Dr. Morse's work is in the second series, or in older children. The prognosis of pyloric obstruction depends entirely on the clinical features, namely, on the degree of obstruction as determined by the amount of constipation, the charcoal test, the degree of emaciation and the strength and the

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persistence of vomiting. If a child is passing a little feces and the body remains stationary, or there is a little gain, we have a right to watch the case. I think the weight, strength and the degree of constipation determine whether or not operation should be performed. Whether the case be one of spasm or of hypertrophy is not important, as complete or incomplete obstruction may be due to either or to both; therefore, I think that a better term than pyloric stenosis would be pyloric obstruction, partial or complete, depending on either hypertrophy or spasm, or a combination of these.

Dr. John L. Morse, Boston: Dr. Sever of Boston is making a large number of roentgenograms of children after administering bismuth in order to get the relations of the intestines. It is not at all uncommon to find a "V"-shaped colon as shown here to-day, and to have the children manifest no symptoms whatever. It does not seem to me that it is justifiable to make a diagnosis of abnormality simply from a picture. If there are symptoms which can be explained in no other way and the picture shows an abnormality, it seems to me reasonable to consider this abnormality as the cause of the symptoms. This is especially true when the putting on of an abdominal band and the giving of exercises relieves the symptoms.

Dr. L. R. DeBuys, New Orleans: There are many chemicals which will give a shadow with the Roentgen ray. I have felt, however, that since bismuth had no ill effect whatever it was safe to use it. There is no doubt that we have in the past been procrastinating too much before referring our patients to the surgeon. They have frequently been given to the surgeon after they have become poor surgical risks. Since the mortality is 50 per cent., it seems to me that we should adopt all the means possible to establish a diagnosis in order to place these cases in the hands of the surgeon earlier. After the diagnosis is made with the aid of the Roentgen ray and we are prepared to turn the case over immediately, if necessary, then we are justified in putting off operation so long as the baby is making a daily gain and improving satisfactorily, because for each day the baby lives, if he gains in weight, his resistance will become greater and he will be better able to withstand an operation.

Dr. Henry Dwight Chapin, New York: With reference to Dr. Cole's question, I consider that possibly due to peristalsis. I cannot say positively. It may have been due to spasm.

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STATUS THYMOLYMPHATICUS

WITH REPORT OF FOUR CASES IN ONE FAMILY

WALTER L. BIERRING, M.D., J. A. GOODRICH, M.D., AND
D. J. GLOMSET, M.D.
DES MOINES, IOWA

The condition that forms the subject of this paper can hardly be referred to as a distinct disease entity, yet it presents such a definite clinical picture, with more or less uniform pathologic findings, that it justifies classifying it as a disease state of special clinical significance.

Its nomenclature is influenced to a certain extent by the age in which the condition is observed, and by the predominance of certain clinical and pathologic features, yet as a whole the terms status thymicus, status lymphaticus, lymphatism and status thymolymphaticus are more or less synonymous. The reference further to the instances of mors thymica and thymic asthma are also closely related to this same condition.

Status thymolymphaticus is peculiar to children, but is also observed in the adult, and is characterized by a hyperplasia of the lymphatic tissues and of the thymus in association with a flabby, fat overgrowth of the body, hypoplasia of the heart and blood-vessels, particularly of the aorta, with a lowered constitutional resistance and a tendency to sudden death from trifling causes.

The four children in one family on whom this study is based, were observed by one of us (Goodrich), and presented the following clinical histories:

CASE HISTORIES

The father of these children is alive and well. The mother died of a heart and lung affection in October, 1910, some eighteen months previous to the death of the first child. In January, 1912, the father again married. In the father's family history there is a record of deaths of eight of his brothers and sisters, all under 9 years of age; three from scarlet fever, one erysipelas, one dysentery, and three from unknown causes. The mother's family history presents nothing of significance.

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CASE 1.—April 8, 1912, Wyetta, the second youngest girl in the family, 4 years of age, died suddenly. For a few weeks some impairment of hearing had been noted, and her breathing had been somewhat difficult and stertorous, but this was ascribed to a cold and was treated with home remedies. About a year previously this child passed through an attack of diphtheria, during which she received 5,000 units of anti-diphtheritic serum. While she recovered very well from the diphtheria infection, she had not seemed as bright or active after this time. During a few days before her death this apathy was more marked. She manifested no desire to play and was greatly disturbed by enuresis, both diurnal and nocturnal. About midnight on the night before her death she aroused her father by an attack of choking followed by vomiting, after which she slept until morning. On arising she was noticed to have a pallid look. She again vomited, was put back to bed, and the physician was summoned, but he found her dead on his arrival.

Necropsy.—Examination made by D. J. Glomset, twelve hours after death.

Record: Length of body 37 inches. The lips are pale. The abdomen is rather prominent. The cervical, axillary and inguinal glands are enlarged. The limbs are plump. The subcutaneous fat is well preserved. The muscles are pale. The peritoneal cavity is free from adhesions. The pleural cavities are free from fluid and there are no adhesions.

Thymus: The thymus is prominent and covers the right auricle completely. It fills the upper part of the anterior mediastinum and lies against the trachea, but there is no evidence of pressure on the windpipe. The organ is made up of two lobes. It measures 5 cm. in length, and 6 cm. in width. It weighs 42 gm.

Heart: The pericardial cavity contains a few cubic centimeters of a serous fluid. The right heart is dilated. The orifices are normal. There is a yellowish-gray nodule on the auricular surface of the anterior lip of the mitral valve. This nodule is about the size of a millet seed. The myocardium appears normal.

Lungs: The lungs are rather boggy, but crepitate throughout. The peribronchial lymph-nodes are enlarged, grayish-red in color and moderately firm. The largest is about the size of a large bean.

Alimentary Tract: Post-mortem changes are present in the stomach mucosa. Six inches above the ileocecal valve the upper part of the bowel has telescoped 2 inches into the lower. There is a hyperemia in the outer part of the bowel, but no signs of exudate or fibrinous adhesions. A large number of the mesenteric lymph-nodes are enlarged, varying in size from that of a pea to a hazelnut. Their consistency is rather firm and their cut surface is grayish in color.

Spleen: The spleen is enlarged, lobulated, and moderately firm in consistency. The malpighian corpuscles are prominent, but apparently lessened in number.

CASE 2.—Eight days following the death of Wyetta, Louise, a bright and active 2-year-old girl, fell down the cellar steps fracturing her left clavicle. This was reduced soon afterwards by Dr. Goodrich. At this time she seemed bright and sat up in bed while the dressings were applied.

The general physical examination revealed a plump, well nourished child. The abdomen was somewhat prominent, liver and spleen not appreciably enlarged. No rachitic phenomena. The tonsils were large, and the cervical lymph-nodes were enlarged. The thymus was not palpable in the episternal notch, yet the percussion area corresponding to its location showed increased dulness. The following morning at about 7 o'clock she arose, and seemed quite well, eating, however, only a little cake for breakfast. Soon after 8 o'clock she was seized

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with a spasm, which was soon followed by another. Then she said she was hungry, and was given some strawberries, of which she ate quite heartily. She was then put to bed and was soon asleep. This did not last long, however, for she was shortly heard screaming. By the time her stepmother reached her she was dead.

Necropsy: Eight hours after death examination was made by Dr. D. J. Glomset.

Record: The body is very pale, a frothy liquid exudes from the mouth, and there are a few ecchymotic spots above the right eye. The cervical and inguinal glands are palpable. A trace of body heat is still present. The abdomen is prominent. Subcutaneous fat is well preserved. The abdominal muscles are pale. The muscles surrounding the left clavicle are infiltrated with blood. The clavicle is broken completely in two, one and one-half inches from the sternal end. The tonsils and adenoids are very much enlarged.

Thymus: The thymus is very prominent, covering the base of the heart and filling the upper and middle parts of the anterior mediastinum. It is composed of two lateral lobes and a central lobe, and measures 4.5 cm. in width, by 6.5 cm. in length. It is rather soft, grayish-white in appearance and weighs 54 gm.

Heart: The myocardium is soft and flabby. The right heart is dilated. There are two small yellowish nodules just above the aortic valve.

Lungs and Trachea: The mucosa of the trachea is congested, but there is no evidence of compression. A frothy fluid exudes from the cut surface of the lungs on pressure.

Liver: The liver is soft and has smooth surfaces. There are two yellowish spots present on the left border of the right lobe which extend about one-fourth inch into the substance of the organ.

Spleen: The spleen is about normal in size. It is distinctly lobulated and the malpighian corpuscles are strikingly prominent.

Stomach: Is moderately distended and contains a semifluid chyme. (This was sent to the chemist for examination, which proved to be negative.) Peyer's patches are prominent and grayish brown in color. The mesenteric glands are enlarged and range in size from that of a pea to a hazelnut. They have a moderately firm consistency.

Brain: Substance is very soft. The surface has a damp appearance. No gross pathologic changes.

Case 3.—June 7, 1912, Walter, the only boy in the family, aged 5½ years, died in the same sudden manner as the two other children. Several weeks prior to this time, he, like his sister, Wyetta, was noticed to have some defect in hearing. On account of this, he was brought to the Children's Dispensary service of Dr. Goodrich on May 20, at which time the examination revealed a boy well nourished, sluggish mentally, having a pasty, adenoid appearance, "pot bellied"; the cervical, axillary and inguinal glands were all palpably enlarged. The tonsils were hypertrophied, adenoids were present. The spleen was moderately enlarged. The lungs were normal. The cardiac area was small. The thymus gland was not palpable in the episternal notch, but a distinctly broad area of dulness was demonstrable in the upper part of the sternum.

The urine findings were negative. Several attempts were made to obtain a blood examination, but were constantly opposed because of the fear of the sim-

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Fig. 1.—Photographs of the four children taken in October, 1911.

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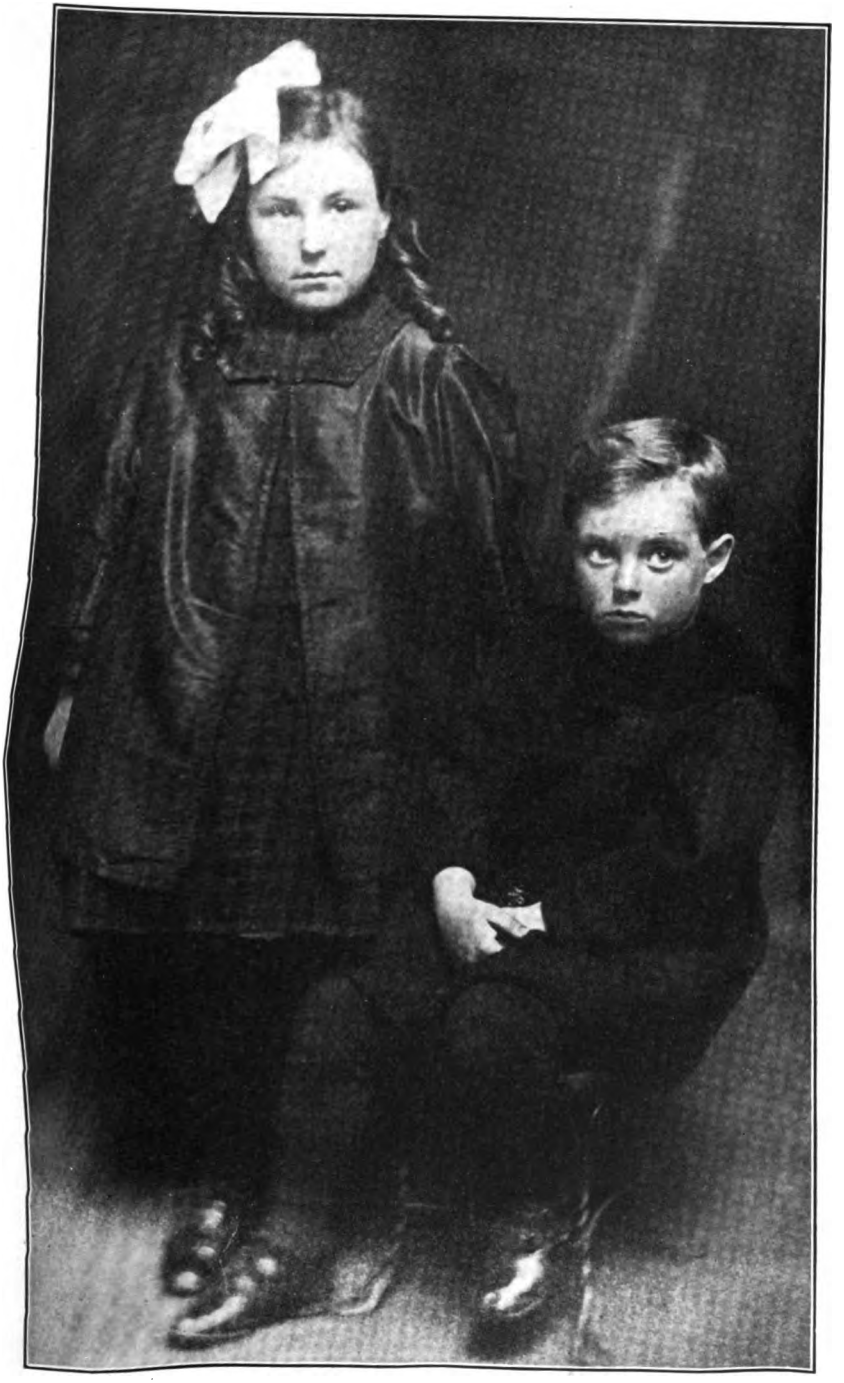

Fig. 2.—The two older children (Cases 3 and 4). Photograph taken in April, 1912.

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ple prick with the needle that was necessary to secure the sample. A similar objection was made to an x-ray examination, or to the suggestion of its use as a therapeutic agent.

The patient was referred to the service of Dr. Pearson for a laryngoscopic and otoscopic examination, but the report was negative with regard to any abnormal findings. The clinical diagnosis of status lymphaticus was made at this time. A course of syrup of ferrous-iodid was prescribed, and the parents were admonished to watch the boy carefully for any change in his condition.

On the morning of the day of his death he seemed to be in his usual health, and had gone out into the garden amusing himself picking potato bugs off the potato bushes, when he was called inside because it was thought the sun was too hot for him. He went into the house and laid down on the bed, but said he was not sick. Soon after this on going to his room he was found to be dead. One arm had been raised over his head and a little bloody froth escaped from the mouth. His face after death, as in the other two children, had a mottled look.

Earlier in the day, this child had experienced some excitement; his sister had returned from school with her promotion card, this being the last day of school, and he became very much interested, so that he could talk of little else than of what he would do when he entered school in the fall.

Necropsy: Made twelve hours post-mortem by Dr. D. J. Glomset.

Record: Body plump, pale, and has a goodly amount of subcutaneous fat. The inguinal and axillary glands are palpable on both sides.

Thymus: The thymus is enlarged, moderately firm and weighs 25 gm. The tonsils are prominent, as are also the collections of lymphatics at the base of the tongue and in the pharynx. There is no evidence of compression of the trachea.

Lungs: There is marked edema of the lungs.

Lymph-Structures: The lymphatic structures of the small intestine are prominent. The mesenteric lymph-nodes are enlarged and moderately firm. The spleen is lobulated and the malpighian corpuscles are distinct. The heart is unchanged. The stomach and its contents were sent to Dr. F. J. Smith, professor of chemistry, Drake University Medical School, for examination as to the presence of any inorganic poison, with a negative result.

Case 4.—July 7, 1912, just three months and four days following the death of the first, the last member of a family of four children, Florence, aged 7 years, succumbed to the same affection.

Since the death of the second child and more specially of the boy, Walter, she had been watched very carefully, not only by her family, but by her attending physician as well. She was examined frequently and presented the same physical changes that had been noted in the other children, as hyperplasia of the lingual and pharyngeal tonsillar tissue, the cervical, axillary and inguinal lymph-nodes. There was percussion evidence of an enlarged thymus, prominence of the abdomen, and a moderately enlarged spleen; the child was rather fat and flabby, had a pasty complexion, but seemed to be in good health. A blood and x-ray examination could not be made for the same reasons as in the other cases.

During the last two weeks of her life the "warning deafness" appeared. On two or three occasions in the week preceding her death, she was seized with spells in which she thought she was being choked, but recovered from them

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rapidly. In one of these seizures she said, "Oh, my God, someone is choking me." She then became cyanotic and sweat profusely; after recovering from it she asked her mother if she had seen all the people about her.

During the night previous to her death she became greatly alarmed because of the thunder storm then raging, but after its cessation she slept quite well. At 7 o'clock in the morning, as her father was going to his work, he awakened her, and as was his custom asked her how she felt; she replied "allright," and returned to sleep. Some two hours later she arose and went to the living room to dress, when it was noted that her head was dropped down on her chest; attempts were made to revive her but without avail and death evidently followed immediately.

Necropsy: Made twelve hours post-mortem by Dr. A. S. Begg.

Record: Body plump, pale, and has a liberal amount of subcutaneous fat. Abdomen is prominent; skin of thorax is mottled and cyanotic. The inguinal and axillary lymph-nodes are enlarged.

Thymus: Enlarged, moderately firm and weighs 40 gm. The dimensions are 4 by 5 centimeters. Marked hyperphasia of tonsils, lymphoid tissue at base of tongue, pharynx, and intestinal tract. Mesenteric nodes are enlarged. Spleen is moderately enlarged. There is some edema of the lungs. Heart is unchanged. No definite pathological change is found to account for death. The possibility of death being due to some chemical poison introduced into the stomach was excluded by a careful chemical analysis of the stomach and its contents after removal from the body.

Microscopic Examination.—The microscopical changes found in the lymph-nodes, spleen and thymus in all four of these cases are very similar and are those which occur in any chronic inflammation of the lymphatic system. There was a diffuse proliferation of the endothelial cells in all of the lymphatic structures. In Case 3 there was also an apparent increase of lymphocytes in some of the mesenteric lymph-nodes and in the thymus. In this case, however, the most marked change was an increase of the fixed cells of the lymph-nodes.

In Case 2, which is fairly typical of the others, the malpighian bodies of the spleen were partially replaced by endothelial cells. Only a few germ centers could be found in any lymph-node. There was a distinct increase in the supporting connecting tissue of the spleen and of the largest lymph-nodes. Giant cells were not to be detected.

In the second case there was also a rather marked degree of myocarditis, and in two of the children there was a diffuse atheromatous change in the aorta of one, and on the mitral valve of the other. Hassall's corpuscles were prominent in all the thymi. There was a homogenous degeneration of the centers of some of the corpuscles of Hassall.

SUMMARY OF CLINICAL AND PATHOLOGIC FINDINGS

1. All the cases presented the same symptom-complex: pasty complexion, a flabby fat overgrowth, hypertrophied tonsils and adenoids, enlarged cervical, axillary and inguinal lymph-nodes and signs of an

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enlarged thymus; all were bottle-fed and had the "pot belly" of the rachitic.

2. Three of the four had a preliminary deafness.

3. Three of the four had seizures of choking or attacks suggesting thymic asthma.

4. All were apparently well nourished.

5. In all no malady sufficient to cause death was noted clinically.

6. In all cases death occurred before the arrival of a physician and under very similar circumstances.

7. No other similar cases are known among the playmates or friends.

8. All were of nervous temperament and became easily excited.

9. One child had diphtheria in 1911 and was given 5,000 units of antitoxin, the other three children receiving at the same time 1,000 units each for prophylactic purposes.

10. The post-mortem findings were practically identical in the four cases: the lymphoid hyperplasia and enlarged thymus prevailed in each. The weight of the thymi were, respectively, 42, 54, 25 and 40 gm.

11. The histologic changes found in all of the cases are extremely suggestive of a chronic inflammation affecting principally the lymphatic tissues. Whether this chronic inflammatory reaction has been produced by poisonous substances formed in some other part of the body or eliminated by some unknown micro-organism affecting the lymphatic tissues, is difficult to say.

12. It is difficult to conceive that the enlargement of the thymus was the main causative factor in the deaths of the children.

GENERAL CONSIDERATIONS

All discussions of status thymolymphaticus center about the conception of the pathological physiology of the thymus, and to a lesser degree of the lymphatic apparatus.

Sudden death has been frequently associated with thymic enlargement, the earliest mention of this fact being made by Plater in 1614.

All consideration of congenital and infantile stridor, asthma and sudden death has given to thymic enlargement, whether associated with

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coincident enlargement of the lymph-node or not, a clinical interest of considerable importance, about which an interesting controversy has been waged for a long time.

Paltauf[1] increased our knowledge of the subject when he described in 1889 a lymphatic constitution; he associated with status lymphaticus a hyperplasia of the heart and arterial system, and ascribed to it the condition of lowered resistance and increased tendency to fatalities in the various acute infections.

In a recent contribution on the pathology of the thymus by Wiesel[2] and the clinical discussion of status lymphaticus as exhaustively considered by H. Matti,[3] a most comprehensive view of the subject is obtained.

The anatomical status of the thymus cannot be said to be definitely fixed. Originally an epithelial structure, these elements gradually undergo various retrograde changes, but the epithelial remnants persist in the connective corpuscles of Hassall. At birth the lymphoid character of the organ is so marked that it is usually classed with the lymph-nodes. Some class it with the ductless glands; others place it without question among the lymphoid organs.

In many ways the thymus may be regarded as a homologue of the tonsils.

The estimation of thymus enlargement is subject to more or less error. Dudgeon[4] places the average weight of the thymus at from 7 to 10 gm. from birth to 2 years of age; Hart,[5], Friedleben[6] and Rolleston[7] also conclude that the acme of growth is reached by the end of the second year, then undergoes retrograde change, yet may remain stationary until puberty, which is followed by rapid physiological involution.

Glands weighing 20 to 30 gm. must be considered as enlarged.

The following tables of weights as collected by three different observers represent the variations peculiar to statistical studies:

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1. Paltauf: Wien. klin. Wchnschr., 1889, ii, 877; 1890, iii, 172.
2. Wiesel: Pathologie der Thymus. Ergebn. v. Lubarsch-Ostertag 15 Jahrg., 1911, Suppl. 2, p. 416.
3. Matti: Ergebn. d. inn. Med. u. Kinderh., 1913, x.
4. Dudgeon: Jour. Path. and Bact., 1905, x, 173.
5. Hart: Grenzgeb. d. Med. u. Chir., 1909, 0, 321.
6. Friedleben: Physiologie d. Thymusdruse. Frankfurt, 1858.
7. Rolleston: Clin. Jour., 1898, xiii.

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Hammer[8]		Friedleben[8]		Von Sury[9]	
Age	Wt Gm.	Age	Wt. Gm.	Age	Wt. Gm.
New-born	13.26	1 yr. 9 mo.	20.7	New-born	14.4
1- 5	22.98	9-24	27.3	1 mo.	15.
6-10	26.10	2-14	27.	2-9	24.3
11-15	37.50	14-25	22.1	9 mo. 2 yr.	23.3
16-20	25.58	25-33	3.1	2-14 yr.	25.
21-25	24.73	.....	....	....	....
26-35	19.87	.....	....	....	....
36-45	16.27	.....	....	....	....
46-55	12.84	.....	....	....	....
56-65	16.	.....	....	....	....
66-75	6.	.....	....	....	....

Hammer's weights are regarded as too high, and were mostly compiled from cases of accidental deaths. Lubarsch[10] claims that status thymicus is very rare in the new-born and young children.

Status thymico-lymphaticus is more frequently observed in older children and adults, thus strongly suggesting that it is an acquired postnatal condition.

The primary cause of thymic enlargement may be sought in any infection, intoxication or disturbance of metabolism, in which there is a lymphoid or myeloid exhaustion. Sahli considers it a criterion of nutrition in infants, associating it with apparently well-nourished pasty children.

The enlargement of the thymus may be regarded, therefore, as a secondary process of the nature of a compensation.

It may be associated with or without lymphatic enlargement. In the same light we are reminded of the observation of Kolisko[11] that the mesenteric glands and lymphoid tissues of the intestinal tract are often so prominently enlarged in enteritis of children as to completely obscure the primary inflammatory process.

In the anatomic findings of the four cases included in this paper, there is revealed principally a lymphoid and fibrous tissue hyperplasia that is very suggestive of a reactive process to some form of irritation.

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8. Hammer: Arch. f. Anat. u. Physiol. Anat., Suppl., 1906.
9. Von Sury: Vrtljschr. f. gerichtl. Med., 1908, xxxvi, 88, Series 3.
10. Lubarsch: Jahreskurse f. Arztl. Fortb., 1912, p. 56.
11. Kolisko: Handb. d. Aerztl. Sarchverst. Tätigkeit, 1906, ii, 701.

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The familial tendency peculiar to our cases has been noted by a number of other observers, Hennig,[12] Friedjung,[13] Perrin,[14] Hedinger,[15] Griffith[16] et al. Hedinger reports an instance in which five members of a family of nine children died suddenly, one of which came to autopsy with the usual findings of status lymphaticus.

Perrin reports in a family of eleven, nine children, and Griffith seven children in one family, from 1 to 8 months of age, who died suddenly with symptoms of dyspnea and cyanosis.

There is no record in the literature of any definite hereditary transmission of this condition.

Our knowledge of the physiology of the thymus is largely based on the experimental work that has been done, principally as the result of thymectomies in animals. The most extensive work is that of Klose and Vogt[17] and H. Matti,[18] from which the following facts are obtained,

Thymectomized dogs present several distinct stages:

1. A latent period of two to four weeks, during which there is no appreciable change.

2. A stadium adipositas continuing two to three months.

3. A stadium cachecticum of three to four months' duration.

4. Death usually within a year.

In addition, a marked change is noted in the general condition, the nutrition is retarded, growth is arrested and bony changes occur very similar to those of rachitis.

There is evidently a distinct connection between thymectomy and the process of ossification.

The removal of the thymus has a depressing influence on the sexual apparatus. A hyperplasia of the thyroid gradually occurs and also some hyperplastic change of the adrenals, hypophysis and pancreas.

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12. Hennig: Gerhard's Handb. d. Kinderkrankh., 1893.
13. Friedjung: Grenzgeb. d. Med. u. Chir., 1900, iii, 465.
14. Perrin: Ann. de méd. et chir. inf., 1903, vii, 217.
15. Hedinger: Deutsch. Arch. f. klin Med., 1905, lxxxvi.
16. Griffith: New York Med. Jour., 1909, xc, 444.
17. Klose and Vogt: Beitr. z. klin. Chir., 1910, lxxix.
18. Matti: Grenzgeb. d. Med. u. Chir., 1912, xxiv, 665.

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In the injection of thymus extract, Svehla[19] has demonstrated a pressor substance. Vincent[20] and Popper[21] report similar results, while Oliver and Schafer[22] and Morehead obtained negative results.

Thymus feeding has not led to any definite results, and the third attempt to produce a state of hyperthymization by means of thymus transplantation, carried out mainly by Sommer and Florken[23] and Ranzi and Tandler,[24] has also not given any reliable or uniform results.

From the experimental results reported, it is evident that dethymization is attended by rather definite systemic disturbances, particularly in the process of ossification and growth of bone, but the opposite state of hyperthymization is by no means as clearly defined.

The apparent association with the chromaffin system is evident, and in the opposing as well as controlling influence of thymus extract on the secretion of the other ductless glands, there is strong suggestion of a separate internal secretion of the thymus, yet it still lacks much corroborative evidence.

In connection with the hyperplastic changes in the thymus and the lymphatic apparatus, some interesting findings have been observed in other organs.

Wiesel refers to the presence of thyroid enlargement in status thymicus. The association of Basedow's disease with status thymolymphaticus has been demonstrated in a large number of instances by Capelle.[25]

The larynx is often small and of the heterosexual type.

The hypoplasia of the aorta, as well as its branches, and the small heart have been referred to. Von Neusser[26] has called attention to abnormal length of the bowel and atypical lobulation of the lungs. Shiota[27] has reported the occurrence of an abnormally long appendix vermiformis.

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19. Svehla: Arch. f. exper. Path. u. Pharm., 1900, xliii, 321.
20. Vincent: Arch. Physiol., 1904, xxx, 16.
21. Popper: Sitzungsber. f. Acad d. Wissensch. Wien., 1905, cxiv, 539.
22. Oliver and Schafer: Cited by Matti, Ergebn d. inn. Med. u. Kinderh., 1913, x; Morehead: The Practitioner, 1905, lxxv, 733.
23. Sommer and Florken: Sitzungsber. d. physiol. med. Gesellsch. Wurzburg, 1908, 3 and 4, p. 45.
24. Ranzi and Tandler: Wien. klin. Wchnschr., 1909, xxii, 980.
25. Capelle: München. med. Wchnschr., 1908, p. 1826.
26. Von Neusser: Zur Diagnose der. Status thymico-lymphaticus, Wien, 1911.
27. Shiota cited by Matti: Ergebn d. inn. Med. u. Kinderh., 1913, x.

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Wiesel[28] has found hypoplastic adrenals in well-marked examples of this condition, and the interesting contribution of Hedinger[29] corroborates the coincidence of Addison's disease with status thymolymphaticus.

In a careful study of fifteen cases of Addison's disease he found eight instances of status lymphaticus, four cases of status thymolymphaticus and three cases incomplete. These observations have been corroborated by Warthin,[30] Pappenheimer[31] and others. Bartel[32] has associated glioma of the brain with the condition under discussion. Marie[33] found acromegaly with status thymicus. Von Recklinghausen reported the presence of status thymolymphaticus in a case of osteomalacia.

The association of a lymphatic constitution with rachitis has been frequently emphasized.

The sudden death in status thymolymphaticus, which forms such a tragic chapter in this condition, has been variously explained, and continues to be a problem of considerable controversy and discussion.

The several theories that have been proposed may be grouped as follows:

1. Mechanical: pressure of enlarged thymus on the trachea or contiguous blood-vessels, causing stridor, asthma and death.

2. Intoxication due to faulty tissue changes similar to the action of syphilis on the heart muscle (Paltauf).

3. Hyperthymization resulting from pathological increase of an internal secretion, both of the thymus and of the lymph-nodes.

Paltauf's explanation has been largely discarded in view of the lack of any supporting evidence.

The mechanical theory is also not sufficient, as so many of the cases show no previous signs of thymic asthma.

Until the existence of an internal secretion has been more definitely demonstrated, it will not be consistent to attribute thymic death to a state of hyperthymization. On the other hand, a critical review of the literature, giving due consideration to all anatomic relations, a mechanical

28. Wiesel: Virchows Arch. f. path. Anat., 1904, clxxvi, 103.
29. Hedinger: Frankfurter Ztschr. f. Path., 1907, i.
30. Warthin: Arch. Pediat., 26, 1909, xxvi, 597.
31. Pappenheimer: Jour. Med. Research., 1910, xxii.
32. Bartel: Wien. klin. Wchnschr., 1908, 783.
33. Marie: Gaz. d. hôp., 1893, lxvi, 202.

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thymus death by tracheal compression, in rare instances also by compression of the blood-vessels, is a distinct possibility, and in a number of instances has been definitely proved to be the cause of death.

Birchner[34] has demonstrated by experimental means the toxic effects of injections with pathologic thymi.

In adults a cardiac death is the rule, and it seems proper to refer to a "thymic heart" in the same sense that the term thyroid heart is used.

Again, it is not so much the thymic hyperplasia as the coincident unfavorable factors that attend this state, which are probably largely to blame for the fatal outcome. The death is often induced by a number of influences that have no effect on the normal individual.

DIAGNOSIS

The recognition of status thymolymphaticus should present no difficulty, since the associated symptom-complex is usually so well defined. Unfortunately, in the past the attention was not directed toward these unfortunate patients until after a sudden death had occurred.

It should be possible to detect complete as well as incomplete types before the fatal exitus manifests itself.

TREATMENT

This resolves itself into prophylactic and curative. Under the former should be emphasized:

1. Avoidance of excitement.

2. No surgical operation unless absolutely necessary, as these patients do not take any kind of anesthetic well.

3. Very warm or very cold baths are to be avoided, as the shock has been known to cause death. Cases of sudden death while swimming may be ascribed to this cause.

4. Prevention of infection, especially of the upper respiratory tract.

5. Securing good hygienic conditions of life.

As regards the curative treatment of status thymolymphaticus, it can hardly be considered as curative in its true sense, since it is more of an attempt to treat and relieve certain symptomatic manifestations of the

34. Birchner: Centralbl. f. Chir., 1912, No. 5, p. 138.

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condition. A medicinal or internal form of treatment is not applicable at all. Of the non-medical therapeutic measures, mention should be made of the following:

1. Intubation with a long tube reaching to the bifurcation of the trachea, being of special value in cases of thymic pressure.

2. Thymectomy, partial or complete, is an operation of considerable surgical risk, yet carried out successfully by Rehn,[35] Veau and Olivier,[36] and more recently by Jackson[37] in this country.

This operation has for its purpose the relief of thymic pressure and removal from the system of the toxic influence of a pathological thymus.

3. Radiation. The use of the Roentgen ray has been espoused by Friedlander,[38] Heinecke[39] and Regaud and Cremieu,[40] and all report brilliant results in producing involution changes in the enlarged thymus. This agent will be equally effective in reducing the size of the spleen and the hyperplastic lymph-nodes.

Because of greater safety and evident efficacy its use is to be recommended.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. BIERRING, GLOMSET AND GOODRICH

DR. C. G. GRULEE, Chicago: I should like to know whether or not there were any marked symptoms of rickets in these four cases. It would be interesting to know whether or not there was any connection between the thymic disease and rachitis.

DR. MARY STRONG, Omaha: I wish to ask if the Wassermann was tried on the other three patients after the first one died. It sounds to me as if some iodid of potassium might have done good in the other three cases.

DR. W. L. BIERRING, Des Moines, Iowa: In answer to the question as to rachitic phenomena, I will say that the patients all had some suggestion of rickets. I was unable to find in the literature any distinct reference to hereditary influence—as to these groups of cases at least. There was, of course, a suggestion in the father's family history of nine brothers having died at an early age, but we were unable to get definite data other than that they had all died before 9 years of age. There is, of course, a strong suggestion that this is a family trait, but when we consider how many instances are on record in which there is status lymphaticus or these allied conditions, without any record of it in other members of the family, it does not seem well demonstrated as yet that there is a hereditary tendency. Of course, we recognize the defect in examination of these cases—that

35. Rehn: Arch. f. klin. Chir., 1906, lxxx, 468.
36. Veau and Olivier: Jour. de méd., March 15, 1912.
37. Jackson: Jour. Am. Med. Assn., 1907, xlviii, 1753.
38. Friedlander: Arch. Pediat., 1911, xxviii, 810, No. 10.
39. Heinecke: Grenzgeb. d. Med. u. Chir., 1905, xiv, 21.
40. Regaud et Cremieu: Thèse de Lyon, 1911.

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there was no examination of the blood and a Wassermann was not performed, but it was impossible after the first death to approach these children with even a surgical suggestion, and we could not even get a roentgenogram picture. Of course the first two children died within nine days of each other. The last two received iodid faithfully without any result. Antitoxin was given in the spring of 1911, and the first death did not occur until April, 1912. One child had a well-marked case of tonsillar diphtheria. This child that had diphtheria seemed more stupid and apathetic during that last year of life, but the other children were apparently as well as they had been.

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VULVOVAGINITIS IN CHILDREN *

RICHARD M. SMITH, M.D.
BOSTON

A striking change has been evident in hospital and medical practice within the last ten years. The advent of social service work in the hospital routine has resulted in a new conception of many diseased conditions. Certain diseases have been long recognized as intimately connected with conditions of living, and steps have been taken toward the prevention of such diseases and the protection of the community from the spreading of the infection. Tuberculosis stands as a notable example of this group. Other diseases until recently considered as presenting almost entirely medical problems are now recognized as having a large social element. As yet sufficient attention has not been given to a study of the social aspect of these diseases. Vulvovaginitis in children belongs to this class. This disease is confined largely to hospital practice, which would at once suggest that it is dependent, in part at least, on poor social conditions. In the Children's Medical Outpatient Department of the Massachusetts General Hospital we have been studying these cases from the social as well as the medical point of view.

The ordinary classifications (Rotch, Holt, Kerley, Koplik and others) of vulvovaginitis in children recognize a non-specific vaginal discharge associated with certain infectious diseases or with local inflammatory processes such as herpes and impetigo. Cases of this kind are not considered here. Another group includes certain

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vulvovaginal infections in which the etiologic organism appears to be some other bacterium than the gonococcus. A few of these cases have been noted in our clinic. The exact bacterial etiologic factor in these cases undoubtedly varies in different individuals. Genito-urinary surgeons recognize certain cases of urethritis in the male which show profuse creamy discharge and are very resistant to treatment, and in which an organism morphologically similar to the gonococcus—the *Micrococcus catarrhalis*—is found. Two of our cases were probably in this group. The third group of cases of vulvovaginitis includes those of true gonorrheal infection. Thirty of these cases have been studied.

The diagnosis of gonorrheal vulvovaginitis is usually made from the finding of morphologically typical gonococci in a stained smear from the vagina. This is open to a possibility of error in certain cases, in that other organisms may simulate closely in appearance the gonococcus. A more definite diagnosis can be made by the means of cultures. The isolation of the gonococcus in pure cultures establishes the diagnosis beyond question of a doubt. The gonococcus is not an easy organism to cultivate. The complement-fixation test[1] is now used extensively as another means of diagnosticating gonorrheal infections. Broadly speaking, the fixation test is like the Wassermann reaction and is specific for the gonococcus. Although there are at present certain atypical reactions not perfectly understood, in general the test furnishes a reliable means of diagnosis, and in conjunction with the other tests of smear and culture it is of distinct assistance in doubtful cases. We have confirmed our diagnosis in nine cases by this means.

The problem of determining the source from which these children acquire their infection presents a real difficulty. Hospital and school epidemics have been

1. Müller: Wien. klin. Wchnschr., 1906, xix, 894. Watabiki: Jour. Infect. Dis., 1910, vii, 159. Schwartz and McNeil: Am. Jour. Med. Sc., 1911, cxli, 693. O'Neil: Boston Med. and Surg. Jour., 1912, clxvii, 464. Smith, G. G.: Am. Jour. Dis. of Chil., 1913, v, 313. Rockwood: Cleveland Med. Jour., 1913, xii, 1.

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recognized and studied for many years.[2] Fortunately at present such epidemics are less frequent than formerly. In these epidemics we are dealing with a known source of infection and have to discover the method of communication from one patient to another within a given institution. This is quite a different matter from determining the source of infection in individual patients who present themselves for treatment in an outpatient clinic. Much difficulty has been encountered in tracing the etiology of these infections. In a report of 344 cases, Hamilton[3] had 45 per cent. of the cases with undetermined sources of infection, and Spaulding[4] in eighty-three cases could not find the cause in 40 per cent. In thirty cases we have been able to determine a possible source for the infection in all but five cases, or 16.6 per cent. One of these undetermined cases was seen at the clinic only three times and was not thoroughly investigated. One gave a history of using a dirty railway water-closet just prior to the appearance of the discharge. One was feeble-minded. In the other two patients no reasonable explanation for the infection could be found. Four gave a history of assault. One was infected in a hospital. In sixteen cases other members of the same family or persons living in the same house were known to be infected. Four cases were of children who contracted the disease from girl playmates. Twenty-one cases, or 70 per cent., were from preventable sources.

SOURCES OF INFECTION

	Number.	Per Cent.
Undetermined	5	16.6
Hospital infection	1	3.3
Playmates	4	13.3
Assault	4	13.3
Other members of family infected	16	53.3
Total	30	....
Known to be preventable	21	70

2. Abt, I. A.: Gonorrhea in Children, THE JOURNAL A. M. A., Dec. 17, 1898, xxxi, p. 1474. Baer: Jour. Infect. Dis., 1904, i, 313. Holt: New York Med. Jour., 1905, lxxxi, 521. Sheffield: Med. Rec., New York, 1907, lxxi, 767. Hamilton: Jour. Infect. Dis., 1908, v, 133. Butler: Interstate Med. Jour., 1910, xvii, 510. Wile, I. S.: Social Plagues and the Public Schools, New York Med. Jour., Sept. 10, 1910.

3. Hamilton, B. Wallace: Gonococcus Vulvovaginitis in Children, THE JOURNAL A. M. A., April 9, 1910, p. 1196.

4. Spaulding, E. R.; Vulvovaginitis in Children, Am. Jour. Dis. Child., March, 1913, p. 248.

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The statement of these causes of infection immediately suggests the social problems in connection with vulvovaginitis in children. Among the children who contracted the disease from some other member of the family, I could find no instance in which instructions had been given about the care necessary to be taken with the discharge. In many instances the infected person did not know that the disease was contagious by any other than the usual method of infection. It is quite possible that in some instances instructions had been given and the family did not choose to follow them, or did not choose to admit that instructions had been given. In any event no care was taken to prevent spreading the disease to other members of the household.

A mother said that her other children had had the same trouble and she "thinks it runs in the family." Another said that an older sister of the patient contracted the disease from a man, but she allowed the two girls to sleep together. A third said that she herself contracted the disease from her husband, but the patient was sleeping in the same bed with the two.

I have not tried to determine the exact manner of infection in these children, but it is probably by direct contact,[5] or by the same means as has been shown to be the method in hospital epidemics, by towels, clothes or other material. Efforts made to close this channel of infection would, I think, show as favorable results here as in connection with the prevention of hospital epidemics. I believe that a considerable number of the infections in our children could have been prevented had proper instructions been given to the persons in the family first infected. I believe that we have a definite responsibility to the community in this connection in making the patient understand the importance of the

5. Abt, I. A.: Gonorrhea in Children, THE JOURNAL A. M. A., Dec. 17, 1898, p. 1474. Cuthbert: Am. Jour. Obst., 1893, xxvii, 698. Westervelt: New York Med. Jour., 1896, lxiv, 748. Schweitzer: Indiana Med. Jour., 1910, xiii, 257.

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disease with which he is infected and the necessity of preventing its spreading to other persons.

The infection contracted from playmates presents another situation which demands the cooperation of a social worker. Bad habits are taught young children by those older than themselves, in some instances by those old enough to understand fully the significance of sexual matters. These habits spread by imitation from child to child. Masturbation is extremely common among the infected patients. Whether as a result of this or not, I am unable to say, but many of these children are sexually precocious. Just how far this has gone in some instances it is impossible to know since their very precociousness makes them secretive. The usual lack of embarrassment during examination seen in a child of 6 has entirely disappeared in many of these children. How far we can go in remedying these conditions must remain for further study; but I am confident that as we learn more intimately the history of infected children, the less often we shall have to attribute the infection to water-closets and other indefinite sources. The supervision given many children by their mothers is so inadequate that there are long periods of time when they are unprotected from the influences of evil companions. We are driven back, in both the family infections and the infections from child to child, to the necessity of educating parents in their responsibility toward their children.

P. was a little girl of 9 years when she first came to our clinic complaining of vaginal discharge. This discharge was proved to be a gonococcus infection. Several days later the mother brought along a small brother aged 7 with urethral discharge, also gonorrhea. The boy's discharge had been present for some time and antedated the appearance of symptoms in the girl. We concluded, erroneously as it later proved, that the boy was probably the source of infection in the girl. After nearly two years of acquaintance with the family the girl made a

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confession of the whole matter to our worker. She contracted the disease by direct contact from an older girl who also taught her to masturbate. She in turn infected her smaller brother.

How important a rôle feeble-mindedness plays in this connection I am not prepared to say, but I am sure that it must be considered as a part of the problem. Feeble-minded children are irresponsible and apparently particularly so in sexual matters. They are often infected with gonorrhea and are ready to pass on the infection to other children. It is extremely difficult to deal with these children in their homes, and the fact that they are a source of direct physical injury to other children makes it even more imperative that they should be removed to institutions especially adapted to their care. In five of our patients the infection is directly traceable to feeble-minded persons, and two other patients were themselves feeble-minded.

A feeble-minded colored child under treatment elsewhere for vaginitis was the inseparable playmate of one of our patients, whose sister also was found to have a vaginal discharge. A mother who is very low grade mentally has one child in the state reformatory and two others coming to our clinic with a gonorrheal vaginitis.

The following family history reveals a situation as pathetic as it is serious: The younger son of a wealthy family in Russia was sent by his parents to this country because he was "crazy." He was unable to earn a living in his own country. After he arrived here he married and became the father of two children. The older of these children, aged 9, is also feeble-minded but not sufficiently so to prevent her being at large in the community. The father insists on sleeping with this child and this child is found to be infected with gonorrhea. She is perfectly familiar with sexual matters and talks freely of the practices of her circle of playmates, who are boys and girls of her own age. Her most intimate friend is a second patient of ours. The habits of the two girls

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are such as to make the passage of infection from one to the other extremely easy. A third member of the group also has a gonorrheal vaginitis. In this train of infections, and undoubtedly others in this neighborhood will be found to be infected, and the original source of infection and the present most active link in the chain should have been removed from the possibility of contaminating others a long time ago. The father's moral influence as exercised through his feeble-minded daughter is quite as injurious as the physical disease which he spreads.

Published reports[4] of the treatment of cases of vulvovaginitis in children have a loud note of pessimism. I do not believe that this is justifiable from the facts. We have been able in our clinic through the cooperation of Dr. Hugh Cabot to have an almost ideal arrangement in the management of these patients. They are all retained as patients of the children's medical department and the home visiting is done by our social workers. The actual mechanical treatment of the diseased condition in the individual patients is done by a member of the genitourinary department who is infinitely more experienced in the treatment of these cases than any man practicing general pediatrics.

The treatment employed varied with individual cases, but in general consisted of a cleansing douche followed by the prolonged application of silver salts. There is some difference of opinion as to what constitutes a cure. Certainly the failure to find gonococci in a single smear, or the temporary absence of a vaginal discharge, is hardly sufficient evidence on which to base a favorable report. It seems to me unfair, however, to refuse to call a patient cured when all symptoms have disappeared and the local examination, including a smear from the depths of the vagina, has been negative for a considerable period of time. If these conditions exist and the complement-fixation test is also negative, I do not see how there can be any question of the cure. I do not agree with those who

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believe that the reappearance of the discharge necessarily means a relapse. When the original source of infection still exists and there has been no reform in the habits of the patient, a reinfection may easily occur and seems to me a more likely explanation than that a relapse has taken place.

The result of treatment in our thirty patients is as follows: One died in an accident before the treatment was completed. In thirteen the outcome of treatment is undetermined because of the failure to continue attendance at the clinic owing to removal from the city or some other cause. Five of these, however, were reported cured and have been free from symptoms for a considerable length of time, although no examination of the local condition was made in the clinic. Eight patients are still under treatment varying in duration to date from one month to two years. It is interesting to note that the patient under treatment two years contracted her infection in a hospital. Eight patients are cured. The cure has been proved by a very long period of freedom from symptoms and local irritation or discharge, and in some instances by a negative complement-fixation test in addition. A detailed table of these cured patients is given herewith:

DETAILS IN EIGHT CASES IN WHICH THE PATIENTS WERE CURED

Case No.	Under Treatment. Months	Without Symptoms. Months	Blood.
3	10	3	Negative (culture negative).
4	7	4	No record.
5	7	3	Negative; previously positive.
8	21	3	Negative.
11	6	6	Negative; previously positive.
18	*	24	No record.
27	5	18	Negative; previously positive.
28	1½	17	No record.

* Time not known; treated by family physician.

The problem of vulvovaginitis in children will demand careful study for many years; but I believe that if we are to make even a beginning toward the eradication of the disease we must recognize that we are dealing not only with a medical disease but also with a social evil, and

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that the medical treatment alone is only a part of what needs to be done. This means that we must establish a close cooperation with the social workers in their attempt to solve the delicate questions of venereal disease. It has been our custom in the children's clinic at the Massachusetts General Hospital to refer all patients with vaginitis to a social worker. We have been particularly fortunate in our social worker. She has been able to gain the confidence of the patients and of their families, and as a result it has been possible to find the probable source of infection in a large percentage of cases. The social records of the vaginitis cases are kept on blanks, a sample of which I am showing, on which the important medical and social data are collected. As has been done for many years with cases of tuberculosis we try to secure an examination of all the other members of the family, at least of all the female children. We instruct the mother or other responsible member of the family in the dangers of infection and we visit in the home to see that our suggestions are carried out. We try to make the parents appreciate their responsibility. In almost every instance when the first infected member of the family was told the true nature of the condition and made to understand the seriousness of the infection it was possible to obtain full cooperation to prevent further spreading of the disease. The instances in which this was not the case were among families evidently on a low intellectual plane or among families generally irresponsible for the welfare of their children. One mother, who had been keeping an immoral house, when she understood the situation moved to a new and clean house and is faithfully doing everything for the benefit of her child. In one instance it was necessary to secure the help of a local board of health to improve the living conditions; but the conditions were improved.

It is possible to find the source of infection in the majority of cases of vaginitis in children. The outlook for cure of the individual patient is good, provided the

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treatment is followed. To prevent the spreading of the disease parents must be made to realize the contagiousness of the condition and the dangers in the neighborhood to which their children are subjected, provided they are not protected during the long hours of play. The frequency with which feeble-minded children act as centers of infection gives an additional argument for their isolation. The treatment of the individual patient as she presents herself for our care remains our first duty, but if we do nothing more we can hardly expect to make much of an impression on the disease as it exists in the community.

VAGINITIS SCHEDULE

Name	Age	S. S. D. No.
Address	Nationality	O. P. D. No.
Date first visit O. P. D.	Date diagnosis	Date referred to S. S. D

I. Information Obtained from Physician

1. Has smear been made — Result
2. Has fixation test been made — Result
3. Has culture been taken — Result
4. Treatment
   a How often is patient to come for treatment
   b Any treatment at home
   c Kind of treatment
5. Local condition — Discharge — Irritation
6. Is patient allowed to attend school

II. Interview with Mother or Attendant at Time Referred to S. S. D.

1. Mother's interpretation of situation before explanation
2. Mother's explanation of source of infection.
3. Social worker's explanation of nature of disease to mother
   a How does mother react
4. Home
   a Other members in family — Infected
   b Any boarders or lodgers — Infected
   c Toilet arrangements — Other families using it
   d Bathing arrangements
   e Sleeping arrangements
5. Recreation
   a How long away from mother's care during day
   b Where does patient play
   c Form of play
   d Playmates — Boys — Girls
   e Reading
6. Habits
   a Cleanliness — What form
   b Hours of sleeping
   c Hours of eating
   d Typical menu — Candy
   e Does patient masturbate
7. Nature of patient
   a Nervous
   b Irritable
   c Obedient
   d Confiding
   e Diffident
   f How much has she learned of sexual matters
   g What are her attachments

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8. Medical social worker's instruction about hygiene
   a Precautions against infection of others
   b Food, bathing, etc., of patient
   c Bringing in of others in family for examination
9. Name of schoolteacher School nurse Physician

III. HOME VISIT
1. Nature of neighborhood and tenement
2. Personal inspection of spleeping and toilet arrangements
3. How is mother following instructions
4. Is confidence of mother and patient gained

IV. VISIT TO TEACHER
1. Patient's school record
2. Playmates, disposition, etc., as noted by teacher

V. SOURCES OF INFECTION
1. Positive Probable Possible

VI. RESULTS OF TREATMENT

329 Beacon Street.

ABSTRACT OF DISCUSSION

DR. FRANK C. NEFF, Kansas City, Mo.: The two points that I have considered to be the least satisfactory in the handling of these cases are the lack of results from local treatment, and the lack of success with the various vaccines, both autogenous and stock. Many of the outpatient children, at least if there is not a sufficient number of visiting nurses, and the child lives at a great distance from the dispensary, cannot get sufficient local treatment to be of assistance. I have seen few patients that one could say, after the lapse of a year or more, were entirely well.

DR. H. M. MCCLANAHAN, Omaha: In my experience those cases are practically hopeless unless they can be followed up. I do not know any stronger argument for social settlement work than this type of case. I have under my care now a little girl in a good family of prosperous people; the mother is cleanly, and I have never witnessed any greater mental anguish than that of this mother when she learned the condition of her little girl. The child herself is innocent. She acquired the disease from a school companion who was a degenerate, was suffering from this disease and insisted on handling the innocent girl. I feel sure that in this case it was the fingers of the infected girl that infected the innocent child. I therefore make the point that the social settlement is one of the modern developments that is needed. I feel that in the families of the poor, to send a child home would be utterly useless unless competent workers could follow up the case in the home. Another point that I have noticed among some of the foreigners in Omaha, one type especially, is that the mothers seem utterly oblivious to the seriousness and importance of this matter.

I have wondered for many years as to the secondary effect of vulvovaginitis in children. I treated a girl sixteen years ago; she was cured and is married now, but she is a sterile

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woman. She is a normal woman and her husband is a normal man and they much desire children, yet she is childless. I believe that she is sterile because she was infected in childhood, although I saw no evidence of pelvic trouble while she was under my care. She is about 24 years of age now, and has been married three years, and we all know that, under normal conditions, a woman married three years becomes a mother in that time.

Dr. H. Lowenburg, Philadelphia: In regard to the therapy of this condition, I must confess that my experience does not permit me to share the optimism of the essayist so far as prognosis goes. I have followed some of these cases at the Philadelphia General Hospital for a period of four and, in a few instances, five years. These patients have been subjected to a variety of treatments at different times. We have tried vaccine, argyrol, permanganate quinin solution, bichlorid and even sour milk, with the idea that the lactic acid bacillus would prevent the growth of the gonococcus. We have secured the same results with all these that we have secured with sterile water. These results have been that after a certain length of time of continuous treatment, except with the vaccines from which we obtained no results, the report from the laboratory would come back negative. Treatment would be discontinued and in two or three weeks there would be a recurrence of discharge containing gonococci. It seems that the effect is a mechanical one and not bactericidal. My experience in the Philadelphia General and Mount Sinai Hospitals has made me come to regard this condition as almost hopeless so far as permanent recovery is concerned.

The point that Dr. McClanahan brought out with reference to sterility in his patient interested me. We may find some explanation in a case I saw about eight years ago in a colored child 6 months of age in the Philadelphia Hospital who was in our vulvovaginitis ward and who died of bronchopneumonia. At necropsy genuine pus-tube was found, from which gonococci were recovered. I do not thing that we have anything in the way of therapy yet that offers a hope of cure in these cases. I think that the greatest hope is in preventive work. In seeking to prevent an epidemic of this scourge in institutions our greatest handicap is caused by what may be called latent cases or cases in the passive or negative stage. These elude the microscopist, are allowed to enter the ward, the disease becomes active again and shortly every other female child is infected.

At the Mount Sinai Hospital we undertook a system of prevention much like the one that Dr. Chapin uses at the Post-Graduate Hospital in New York, and yet cases of vulvovaginitis arise in wards after every new patient is kept in the observation ward for three days and three negative cul-

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tures are obtained. I think that the reason is, as just stated, that they are received in the latent stage, wherein the discharge is absent or appears normal by examination. In a few days or a week, when it is too late, its infectious nature is made manifest. I have been embarrassed many times when children were brought to the hospital with no discharge, suffering from typhoid or pneumonia, recovered from that and went home with a vulvovaginitis. Sometimes I feel that it would have been better had they succumbed. This is a dangerous condition, and we have not yet found a cure or a certain prevention for it.

Dr. D. L. Richardson, Providence, R. I.: I have nothing in the way of treatment to offer. My own efforts have been as fruitless as those of others. In the question of control, particularly in hospital management, there is work to be done both by the hospital and the social workers. Since it is very difficult to cure these cases permanently, so long as they are active and there is a good deal of discharge, the hospital is the place for them. During the periods of remission they are of less menace at home. It is customary in many hospitals, particularly children's hospitals, to take smears on admission and any suspected case is refused admission. In our contagious hospital we take smears in every case, no matter what the disease, in children under 15 years of age. If any case is suspected, it is isolated throughout its hospital residence. I do not think that it is safe to depend on any number of negative smears. Once infected I think that it is always infected so long as it is likely to remain in the hospital. It can be isolated in any hospital as well as a contagious hospital. It is a question of careful technic. It is not sufficient to put all cases in a side room and depend on that to control the epidemic. It means that everything that comes in contact with that patient must be free from infection before it leaves that room. We have had a good many cases of vulvovaginitis in our infectious wards, but never any transmission. The nursing must be absolutely careful and correct as to technic. For that reason I say any general hospital with accommodations, running water, etc., can care for these cases as well as an infectious hospital can.

Dr. Frank C. Neff, Kansas, City, Mo.: May I add one word in regard to prophylaxis in the management of the child at birth? Undoubtedly many of these cases begin at that time and should receive the same silver treatment, or other treatment, as the eyes.

Dr. Julius Hess, Chicago: I cannot say that I share the pessimism of Dr. Lowenburg. I should like to ask Dr. Smith as to whether the application of silver is vulvar or both

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vaginal and vulvar. There has been much discussion as to the advisability of vaginal application.

Dr. C. F. Wahrer, Fort Madison, Iowa: The great question is how these infections come among children when the parents themselves maintain that they have lived pure lives. I think that people are not clean enough. You will notice how much attention animals pay to their sexual apparatus. It is surprising how very little attention mothers of children pay to the sexual apparatus of their children. I have seen children of five years with gonorrhea. I had to convince some of these parents by making smears and showing them by illustrations the microscopic field, and even then one mother became very indignant. I do not share the extreme pessimism of Dr. Lowenburg, but we all have our individual experiences and we sometimes depend on these experiences. His experience warranted him in saying what he did. With the few cases of vulvovaginitis that I was able to keep under my care I have been singularly successful. We have few hospital facilities for such cases. They are not received in the hospital for fear of contaminating the surgical ward. I believe that, if we cannot have the social settlement worker, the doctor himself must be the social settlement factor and teach the parents how easy it is for these contagious diseases to be spread. Your little girl, a pure child, plays with another who is not so pure, and she plays with others less pure still, and this is a means of handing these infections about. These children have eczema and other irritations and scratch themselves and handle the playthings of other children and then the hands of these children are infected. When we know how gonorrhea is carried about in the usual manner it is a great factor in the social world (a social disease), and when it comes to the little children in this indirect manner it is a serious thing.

Of course sterility, such as Dr. Smith mentioned, can happen in a child as well as an adult. I think that we should give persistent treatment with silver preparations, medicated bougies, etc., if we are to keep control of cases that leads to success.

Dr. John Zahorsky, St. Louis: I am not so pessimistic about the result of these cases. I have noticed a difference in hospital cases and cases in private practice. I do not think that recovery takes place so quickly in hospitals as in private practice. I believe that the child who stays for a prolonged period in a hospital acquires a lessened resistance. After all, gonorrhea is cured not by antiseptics, which reduce the number of germs, of course, but by some sort of local or general immunity established by the child itself. Unless this immunity is established there will be a continual recurrence and reinfection, because a few germs will escape

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somewhere in the folds of the mucous membrane. The vaccines have proved a failure in this direction. We still hope that some vaccine will be found that will assist the patient in establishing a local or general immunity. At present we have to depend on a good, healthy resistance of the child. That is not established in hospitals. Children in hospitals with lessened resistance and those in poor home surreundings will not give as good results as those in good homes and in the country. Some children have no resistance to any infection. As a rule, in healthy families and healthy surroundings, if the child receives good general attention as to diet and exercise which keeps it in the best possible condition, with local assistance of cleanliness, it will recover.

Dr. H. D. Chapin, New York: There is no subject which has caused some of us so much distress, particularly in institutional work, as this. Dr. Lowenburg mentioned a type of case that has occurred with all of us—it certainly has with me. A little girl is sent into the hospital for some illness, and contracts vulvovaginitis. I cannot imagine anything more distressing than this. I should like to tell you what I have done, so far as institution work is concerned. We take smears, of course, as I suppose is done in most cases. The strange thing is that many of these children after having been in hospital for several weeks show a discharge. In many instances there has been no other case of the infection for a long period of time. I have never been able to understand why that particular child should develop this condition at that time, but probably the gonococcus was latent. It is undoubtedly spread by diapers. We used every method we could think of to sterilize the napkins, and yet the disease occasionally spread. We now use a different system. Of course, this is limited to girls, as you know. Every little girl wears a vulvar pad from her entrance until the time she leaves the hospital, and we do not use ordinary napkins. We use cheese-cloth entirely. When taken off, the chees-cloth napkin is put in a paper bag, the bag sealed and the whole thing is burned. Every child has an individual thermometer, etc. In this way we have succeeded fairly well in keeping the wards clean. It is a little more expensive to do this, but no trouble and expense is too great for avoiding even a single case of this terrible disease.

I am not so pessimistic as Dr. Lowenburg with regard to local treatment.

I was much interested in the case mentioned of an infant 6 months old with a pus-tube. I have seen one case in a little girl of three, who had to have a laparotomy and the tube removed. Outside of these cases, I have never known of the gonococcus involving these deeper tissues in children. The eye is seldom infected in my experience. Whether this

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is due to a different strain of gonococcus, or increased resistance, I do not know. We all know how hard it is to keep children's hands away from their eyes. One of my nurses in the Willard Parker Hospital once lost an eye. I had a symposium on this subject at the pediatric section of the New York Academy of Medicine last winter. The best men were there. I was cheered by one thing—several of our observers who have had a wide experience said that they knew many cases in which these little girls had grown up and had borne children, and had apparently not been ultimately injured by this disease. Several also said, and I must say it has been my experience, that the disease seems to wear itself out—a spontaneous cure takes place.

Dr. R. M. Smith, Boston: I realize that in the limitations of this paper it is impossible to mention all points. The question of hospital epidemics presents a wide field for thought. We have been particularly fortunate in our hospital in this respect. We use ordinary precautions and thus far have had no epidemics.

I am not surprised at the pessimism expressed about the cure of this disease. I think a good deal of this pessimism is due to recurrences which are called relapses. If a child contracted the original infection by a certain means, and that means still exists in the community, there is no reason why the child should not get a fresh infection. I believe that many children do.

I did not say much about treatment in my paper. It is a problem in itself.

There was variety in the kind of treatment which our patients received. They were all given a long douche, sometimes with permanganate, sometimes with boric acid, till the water comes back perfectly clear. Then an application of some silver salt was made. This local application was retained for a considerable period of time. After the preliminary douche, several ounces of the silver salt solution were injected into the vagina, the hips were raised and the child left in that position for at least half an hour. Then a vulvar pad was applied and as much as possible of the solution retained. The longer it stays in the better. This treatment is repeated as often as is indicated by the individual patient. It is important to continue the treatment for a considerable time after the patient apparently has no discharge. The patient should report for observation once a week for a time and then twice a month, and then once a month for a period of years.

In taking smears, it is important to go into the depths of the vagina. A superficial smear does not amount to anything.

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I did not mention complications. It has been mentioned that pus-tubes occur. That is well recognized. In our clinic in the last two years we have seen two acute cases of pus-tubes. We have seen one case of arthritis. One child with pus-tubes was about 5 years old, the other was younger. The child with arthritis was about 7. I do not know anything about the question of sterility following tubal infection.

We have had no success whatever with vaccines.

I am sure that a large proportion of these cases of infection come directly from handling by themselves or by other children. We encounter a great many experiences in these girls similar to that mentioned by Dr. McClanahan. If one secures the confidence of these children they will eventually tell the story. In practically every instance we have found that the habits of the children were such that infection was easy.

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RUPTURE OF A MEDIASTINAL LYMPH-NODE INTO THE BRONCHUS

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History.—G. S., aged 5, was admitted from the German Hospital Dispensary to the A. Jacobi Department for Children, Nov. 25, 1912, with a diagnosis of pus in the urine. The child had been in remarkably good health; the birth was normal; the baby was breast-fed for two years. Until the present illness life was uneventful; the child had no acute or chronic illness, no coughing or expectoration, and no gastro-intestinal complaint. No tuberculosis is present in the family of the father or mother. The mother had no miscarriages. The two living children are in perfect health. Four months before, the mother noticed mucus in the urine, and the child complained that urination was painful. He cried when urinating, but there was no increased pain at the end of urination; neither was there increased frequency. A few weeks before the mother noted a slight trace of blood in the urine.

Physical Examination.—The patient is a well-nourished little boy; pupils are equal and react to light; no swelling under the eyes. The cervical, axillary and inguinal lymph-nodes are normal. The tonsils are hypertrophied; the throat otherwise is negative. The heart percusses within normal limits; the sounds are clear; no murmurs are heard. Percussion and breathing-sounds show the lungs to be in normal condition. There is no tenderness over the suprapubic region, over the region of the kidneys or over other parts of the abdomen. Percussion of the liver shows that it extends nearly to the costal border; the edge is not felt. The percussion note over the spleen is normal. There is no tenderness of the region of the kidneys; neither kidney can be felt. Both testicles have descended and are normal. The extremities are in normal condition; knee-jerks are present; there is no edema.

Laboratory Reports.—The urinalysis showed a faint trace of albumin. The urine was yellow and turbid, with a white mucoid sediment. The centrifuge specimen showed few blood-cells, many leukocytes, and few triple phosphates; no tubercle

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bacilli were found, but the culture showed a growth of colon bacilli.

Examination of the blood showed conditions normal. Cystoscopy revealed ulceration on the right side, near the trigon. Trabeculae were seen in several places over the bladder wall; the meatus of the right ureter was enlarged and reddened; the urine from the ureter seemed to be clear. Dr. Rehling, who did the cystoscopy, thought that the ulcerations were not of a tuberculous nature, and no definite diagnosis was made. Roentgenoscopy of the genito-urinary tract on both sides did not reveal the presence of nephrolithiasis or other abnormality.

Treatment and Course.—Treatment consisted of regulating the diet and giving small doses of hexamethylenamin (urotropin) three times daily. With rest in bed, the urine became clearer from day to day and the boy was apparently doing well. On the night of December 5, eleven days after his admission to the hospital, he was put to sleep with the other children in the ward after having partaken of the usual evening meal, feeling perfectly well and in good spirits. About 11 o'clock at night the nurse, on going from bed to bed, noticed that the child was somewhat blue and was breathing heavily, and, on trying to arouse him, found that he was unconscious. She at once notified the house surgeon, who came to the bedside finding the child with flushed face, dilated pupils and rapid breathing. It was first thought that the child was suffering from some drug poison, possibly belladonna. I was summoned to the hospital and found the child cyanotic, pupils dilated, head thrown back, arms drawn up, legs spastic, big toes dorsally flexed, and knee-jerks exaggerated. Drug intoxication was excluded.

The stomach was lavaged; small flakes of food particles were returned, and lavage was continued until the water returned clear. The bowels were thoroughly irrigated; a lumbar puncture was made, and the fluid returned under slight pressure. A tentative diagnosis of tuberculous meningitis was made; the head was elevated, an ice-bag was applied, and the child was given 2½ grains of calomel. I returned home and the house staff went to bed. About three hours later the house surgeon was again summoned hurriedly to the bedside of the boy and found him apparently in collapse and lifeless. The face was almost black. Artificial respiration was resorted to but with no result. Intubation was then performed, which was also without result. Then a tracheotomy was done, but the child did not respond on the introduction of the tracheotomy tube. A catheter was introduced through the tracheotomy tube into the trachea, but without effect. Then the insufflation apparatus was connected with the end of the catheter and, when the catheter was moved back and forth, suddenly from half an ounce to an ounce of a thick cheesy mass exuded from the tracheotomy tube, between it and the catheter. On the

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expulsion of this thick mass the child began to breathe more freely, the lips reddened and the cyanosis disappeared. Although the general appearance of the child was greatly improved, he remained unconscious for four or five hours longer, when consciousness gradually returned.

During the next twenty-four hours his condition remained about the same, except that he occasionally dropped into a stupor-sleep. During the first day after the tracheotomy he vomited twice, the vomitus consisting of some undigested and unchewed Lima beans which he had eaten the day before and which were too large to have been removed by the lavage the night previous. The temperature during his entire stay in the hospital and for the first day after the tracheotomy remained perfectly normal. The pulse-rate was also within normal limits.

The day following—that is to say, twenty-six hours after the tracheotomy—the child developed an inspiration pneumonia, which ran its regular course. In the excitement of saving the child's life, the pus or cheesy mass which was forced out of the trachea was lost, with the exception of a small quantity which remained on the dressing, a culture of which showed a growth of streptococci.

The third day, with the exception of the bronchopneumonia, the child's condition was very good. The knee-jerks were present and were not exaggerated; the Babinski reflex was negative; the Oppenheim reflex was not obtained. Kernig's sign was practically within normal limits. There were no paralyses.

December 9 the eyes were examined by Dr. Denig and a slight indication of choked disk was observed. The ears were examined and found negative. The laboratory report of the spinal fluid showed a negative result from the globulin, albumin and Fehling tests. The cell-count showed 8 cells per cubic centimeter. No tubercle bacilli were found, and the guinea-pigs which were injected with spinal fluid have never showed any symptom of tuberculosis. The blood-count made the first day after the tracheotomy showed white blood-cells, 21,800; polynuclears, 70 per cent.; lymphocytes, 21 per cent.; small mononuclear lymphocytes, 9 per cent.; the blood-culture was sterile. The stools were lumpy and yellow, showing few curds. No parasites were found.

Since the possibility was realized that a rupture of an abscess or lymph-node into the trachea might have caused the symptoms, the child was sent, December 6, one day after the tracheotomy, to the Roentgen-ray department for roentgenoscopy. The report was as follows: "A somewhat widened shadow at the base of the heart. This is most marked on the left side of the spinal column and may indicate some involvement of the mediastinal lymph-nodes. On the left side, at the root, is seen a small amount of consolidation. On the right

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side of the spinal column, just above the shadow of the right heart, is seen protruding a circular faint shadow; this is most marked in the fourth intercostal space, and behind and above it in the third interspace is also another faint circular shadow. These usually indicate enlargement of the mediastinal lymph-nodes, and associated with a clinical history of a bladder condition I feel certain that we are dealing with a broken-down lymph-node which either has ruptured into the bronchus or was producing asphyxia by constricting the trachea, and the slight deviation of the trachea to the right just above the bifurcation rather points to this condition. If this is so, then the end of the tracheotomy tube or, rather, the catheter, probably ruptured the broken-down lymph-node abscess-cavity."

In a review of the literature in search of similar cases, the first that came to my notice was the one reported by Dr. H. D. Chapin[1] as a case of sudden death from pressure on the pneumogastric nerve, with necropsy. This was a patient in the outpatient department who was brought in for a slight cough. The only abnormal finding in the examination of the child was a slight difference of breathing on inspiration, as if there was a narrowing of some canal in the respiratory tract. During a number of weeks while the child was under observation, repeated examinations showed no change in its physical condition. One day the mother came to Dr. Chapin and reported that the child had died the night previous. At 6 o'clock on the evening of that day the child ate a hearty supper, was in good spirits, and was laughing and playing when at 8 o'clock it was seized with a paroxysm of coughing, dyspnea with marked cyanosis, and sudden death. The necropsy revealed enlarged mediastinal lymph-nodes with a ruptured abscess-cavity pressing on an inflamed pneumogastric nerve.

F. Petersen,[2] reported a case of obstruction of the trachea through a caseated and softened bronchial lymph-node, cured by means of tracheotomy. The patient was a girl aged 6, on whom tracheotomy was performed on account of severe dyspnea, with facial cyanosis and unconsciousness. Respiration was not much improved

1. Chapin, H. D.: N. Y. Med. Jour., March 15, 1884.
2. Petersen, F.: Deutsch. med. Wchnschr., 1885.

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by the tracheotomy, but the correct diagnosis became apparent when small amounts of cheesy débris were found on a catheter which had been introduced into the trachea and when numerous large and small particles were forced out by the coughing until a large caseated and calcified piece of lymph-node was expectorated. Respiration then became free, and the child was discharged well one month later. No tubercle bacilli were discovered on examination of the material.

F. Nachod[3] reported a case of bronchial stenosis due to rupture of peribronchial lymph-nodes into the air-passages of a boy aged 6½. The diagnosis of obstruction of the right bronchus had been made before the performance of the tracheotomy, which served to give exit to large pieces of chessy lymph-node from the right bronchus. The patient recovered after diffuse bronchitis and right-side pleurisy. Microscopic examination of the eliminated masses showed them to consist of lymph-nodes almost entirely caseated. The examination for tubercle bacilli had a negative outcome.

E. Gerber[4] observed a case of asphyxiation due to rupture of the caseated lymph-node into the trachea. At the necropsy a caseated lymph-node was found to have blocked the main bronchus. Tuberculous peribronchitis was demonstrated in addition to the perforation. The patient was a boy, aged 6, who was suddenly attacked with asphyxia and was dead before he reached the hospital. Death was due to the perforation of the anterior wall of the trachea, and to the passage through the opening of a piece of cheesy lymph-node which blocked the air-passage. The left lung was free, but several tuberculous foci were found in the right upper lobe. Gerber assumes that the condition causing death developed insidiously by the cheesy lymph-node first infecting and then disintegrating the tracheal wall.

3. Nachod, F.: Prager med. Wchnschr., 1897, No. 33, p. 393.
4. Gerber, E.: Deutsch. Ztschr. f. Chir., 1904, xcv, 361.

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V. Gandiani[5] reported a case of rupture of a caseated lymph-node abscess into the trachea. The patient was a child aged 3, who three days prior to admission became ill with cough, fever and respiratory disturbances. The pharynx was reddened, and the tonsils presented small isolated necrotic foci. The general condition was bad. Diphtheritic serum was administered. The child had a sudden attack of dyspnea with rapidly progressive cyanosis and symptoms of asphyxiation. Tracheotomy was performed, but the child died half an hour later. At the necropsy the trachea was found to be surrounded by a large packet of lymph-nodes as far as the beginning of the bronchi. The anterior wall, a few centimeters above the bifurcation, presented an orifice the size of a lentil. Through the perforation in the trachea a fairly large cavity was reached, which was emptied by pressure from outside, and contained tough, cheesy masses, which also blocked the bronchi. The peribronchial lymph-nodes were tuberculous.

Loehrer[6] reported the case of a boy, aged 4, who suddenly died under the signs of acute asphyxiation without having been ill in any way. At the necropsy a grayish-yellow structure of rather firm consistence was found which solidly packed the lumen of the trachea, filling it almost up to the bifurcation. The right bronchus, moreover, contained an opening 8 mm. in width through which a cavity about the size of a walnut, with rigid walls, was reached. This cavity was filled with pasty, friable masses. Animal experimentation showed that the changes of the lymph-nodes were due to tuberculous caseation. Loehrer points out that the softening of the caseated tuberculous lymph-nodes of the neck and the bronchi, and especially their rupture into adjacent hollow organs, are probably the result of a mixed infection.

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5. Gandiani, V.: From the San Giovanni al Saterano Hospital, Rome, Deutsch. med. Wchnschr., 1904, No. 24.
6. Loehrer: München. med. Wchnschr., 1904, No. 27, p. 205.

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Biedert and Fischl[7] speak of lymph-nodes without tubercle bacilli or other evidences of tuberculosis, and state that this is probably a very rare occurrence.

Skala[8] reports the case of a girl of 14, on whom a tracheotomy was performed on account of rapidly progressive dyspnea. Opposite to the tracheotomy wound in the membranous posterior wall of the trachea was a constricting abscess, the pus of which contained streptococci. The etiology of the abscess—whether due to a foreign body or originating from the adjacent lymph-nodes—was doubtful. The operation was followed by prompt recovery.

C. Comba[9] observed an unusual complication of tuberculous peribronchial lymph-nodes in a child. The boy, aged 11, had a dry cough, swelling on left side of neck, and dyspnea. On his admission to the hospital, bronchial breathing and crepitation were heard on the left side of the thorax. Three weeks later the patient died under symptoms of tuberculous meningitis. At the necropsy numerous swollen lymph-nodes were found in the anterior and posterior mediastinum. The bronchus presented an ulceration. Evidently a large lymph-node had been pressed against it by the mass of mediastinal lymph-nodes, with the result that the bronchial wall had been perforated. A fragment of caseated gland had then penetrated into the bronchus and entered into the trachea, causing severe attacks of dyspnea.

Rabot and Di Villiers[10] observed a boy who had attacks of paroxysmal coughing which resembled whooping-cough. Under progressive aggravation of the condition the patient died at the end of two weeks. The necropsy showed numerous lymph-nodes along the trachea at its bifurcation and around the large bronchi. The right lung contained a cavity. Several punctiform tubercles

7. Biedert and Fischl: Lehrbuch der Kinderkrankheiten, 1902.
8. Skala: Wien. klin. Rundschau, 1904, Nos. 41-42.
9. Comba, C.: Rev. di. clin. Pediat., April, 1905, p. 241.
10. Rabot and Di Villiers: Lyon Méd., 1905, No. 19.

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were also found at the base of the lung. Microscopic examination confirmed the diagnosis.

Knoepfelmacher[11] demonstrated an anatomic specimen in the case of a child, aged 2½, who had been under treatment eight days for diphtheria. Tracheotomy had been performed for sudden extreme dyspnea. The trachea was found to be free, but the dyspnea and pulmonary emphysema persisted. The necropsy confirmed the suspicion that the right main bronchus had been blocked by a tuberculous lymph-node.

Sternberg[12] reported before the Brünn Medical Society the case of a child, aged 2, who died suddenly after the rupture of a caseated mediastinal lymph-node into the trachea. The point of rupture was at the bifurcation. The trachea contained an oval piece of a caseated lymph-node about 2 cm. in length.

C. Ebert[13] reports three cases of rupture of mediastinal lymph-nodes into the trachea, with necropsies. In only one of these cases was the tubercle bacilli demonstrated.

E. Dina[14] contributes an observation on perforation of the air-passages by caseated bronchial lymph-nodes. The patient was a child, aged 5, who was admitted to the clinic with severe asphyxiation. The condition suggested laryngeal diphtheria, for which intubation was performed. Respiration still remained difficult and did not become normal until a yellowish-white mass was expelled in a coughing-fit from the tracheotomy wound. On examination, this body was found to be a caseated lymph-node in which tubercle bacilli could be demonstrated.

Coakley[15] reported a death through suffocation due to pressure of a tuberculous mediastinal lymph-node on the trachea. The patient was a girl, aged 3. Necropsy showed an oval mass, 2.5 mm. in length, lying under-

11. Knoepfelmacher: Wien. med. Wchnschr., 1907, No. 23.
12. Sternberg: Wien. klin. Wchnschr., 1907, No. 29, p. 900.
13. Ebert, C.: Internat. Centralbl. f. Laryngol., 1911, xxvii.
14. Dina, E.: Rev. di clin. Pediat., November, 1909.
15. Coakley: Tr. Am. Laryngol. Assn., Washington, May, 1910.

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neath the tracheal mucosa and entirely blocking the lumen. A tuberculous focus was found in the left pulmonary apex, and also tuberculous peritoneal lymph-nodes. D. Bryson Delavan also observed a similar case.

Maillet[16] pointed out that certain symptoms in the form of acute functional disturbances may suddenly appear in the course of tracheobronchial adenopathy and may actually represent the first manifestations of a previously unrecognized or latent adenopathy. These symptoms may be of a grave, or at least alarming, character. They may be divided according to their involvement of the respiratory, circulatory or digestive apparatus. The respiratory symptoms are the most common and also the best known. Two illustratives cases are given—one, in a girl who had had a severe coughing fit with extreme dyspnea; the other in a boy, aged 2, who suffered within a week from two attacks of dyspnea, with cyanosis of the face. These attacks of acute respiratory tracheobronchial adenopathy must be differentiated from the attacks of dyspnea due to hypertrophy of the thymus.

Oeri[17] reported a case of rupture of a bronchial lymph-node into the bronchus, with a favorable outcome.

Ubert[18] reported a case of tracheal stenosis from a caseated, calcified and softened bronchial lymph-node. The patient was saved.

H. Grenert[19] gave a short review of this form of tracheobronchial adenopathy.

Betke[20] reported a case of bronchotracheal stenosis due to tuberculous lymph-nodes at the bifurcation in a woman, aged 29, who was cured by surgical interference (longitudinal mediastinotomy, according to Sauerbruch and Schumacher).

From roentgenoscopy of the chest of all children admitted to the A. Jacobi Department for Children, I am convinced that the existence of enlarged mediastinal

16. Maillet: Ann. de med. et. chir., inf., 1911, xv, 511.
17. Oeri: München. med. Wchnschr., 1913, No. 8, 410.
18. Ubert: Inaugural Dissertation, Kiel, 1885.
19. Grenert, H.: Ann. di med. et chir. inf., 1911, xv, 479.
20. Betke: Berl. klin. Wchnschr., 1913, No. 4, p. 197.

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lymph-nodes is more frequent than one has been led to suppose. Without roentgenoscopy it is, in the great majority of cases, absolutely impossible to make a diagnosis of their existence by either auscultation or percussion. It may be true that a great many persons with enlarged mediastinal lymph-nodes grow to adolesence and adult life without showing symptoms of the condition, but it seems to me that when their existence has been demonstrated this condition should be borne in mind and the child treated accordingly. From a review of the cases just cited, it is evident that the onset is usually very sudden; often, as in the case which I have presented, in an otherwise apparently healthy child. The symptoms may be divided into those showing involvement of the respiratory, the circulatory or the digestive apparatus, or a combination of all three. Those of the respiratory type show either slight respiratory sounds in auscultation, slight cough, or more marked labored breathing. The cases of the circulatory type show more or less cyanosis, accompanied with a slight respiratory cough or none at all. The digestive type starts in with fever, vomiting followed by difficult breathing, cyanosis, unconsciousness and sometimes death. These functional symptoms are varied—sometimes pronounced and sometimes practically absent. In quite a few instances when the digestive type of symptoms is presented there is also marked meningeal irritation, as in the one which I have given, and from the symptoms which the child presents it is impossible to differentiate between this form of meningismus and a true meningitis. The more I see of these cases the more I am impressed with the fact that a great many of the cases of so-called gastro-intestinal disturbance with meningeal symptoms are often cases of mediastinitis with local suppuration and absorption, rather than true intestinal toxemia. It is evident from the frequency of these enlarged mediastinal lymph-nodes that they no doubt stand the brunt of the battle of bacillary pulmonary invasion.

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As regards the etiology, one must divide these cases into those of tuberculous and non-tuberculous infections. The non-tuberculous infections may be subdivided into the true streptococcus or the true staphylococcus, or a mixed infection of the two.

In the case which I have reported, repeated examinations to demonstrate the existence of the tubercle bacillus has been unsuccessful. I have the boy back again in the hospital and have injected guinea-pigs with catheterized urine obtained from the patient's ureters, and up to the present writing the guinea-pigs have shown no evidence of tuberculosis.

The necropsies in all the cases show about the same findings. The enlarged and suppurating lymph-node or lymph-nodes either press forward and partially obstruct or totally occlude the bronchus and trachea, or they press backward on the pneumogastric nerve, thoracic duct or some vessel situated in the immediate vicinity of the mediastinum. In fact, I have seen cases at necropsy in the hospital in which the mediastinal lymph-nodes have perforated into the adjacent parenchyma of the lung and formed a localized abscess which produced absolutely no symptoms *intra vitam* and was discovered only at the necropsy.

As to differential diagnosis, these cases must be distinguished from: (1) the true type of meningitis in all its forms; (2) from cases of suspicious cough or pressure on the main branches of the air-passages when the usual causes of such conditions can be excluded, and (3) from the so-called intestinal toxemias of childhood of the mild form, or of the graver form accompanied with meningeal irritation, high fever and cyanosis.

The prognosis depends entirely on whether the lymph-nodes remain in a simple enlarged state without producing any pressure on the adjacent organs, whether they go on to suppuration and subsequent perforation, or a combination of all three conditions, the most favorable

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course being, in case of suppuration as indicated by the many cases reported, perforation into one or other of the larger main branches of the upper air-passages and expulsion either by fits of coughing or by prompt surgical interference, as in the case cited.

The treatment of these cases resolves itself into medical and surgical. The medical treatment, as soon as the diagnosis is made, should consist in the same precautions as are indicated in other forms of incipient tuberculosis. These cases in which the lymph-nodes are hidden in the mediastinum do not differ from those cases of tuberculosis of the lymph-nodes of the neck which can be seen and which require constant attention. The child should sleep in a well-ventilated room, and should be given plenty of pasteurized milk and a good wholesome diet suited to its age, with the continuous administration—not for weeks or months, but for years—of small doses of guaiacol and arsenic. Of course, when these cases call for surgical interference, this should be promptly rendered. With the improved method of mediastinal surgery by the aid of the Sauerbruch chamber or the Meltzer-Auer apparatus, it will remain a question for future obesrvation whether or not, in cases showing symptoms of slight, moderate or more severe forms of mediastinal obstruction, it is advisable to open the mediastinum by a median mediastinotomy and remove the lymph-nodes.

I wish to thank Dr. Sanford, the house surgeon, who by his prompt and persistent measures and surgical skill in the case which I have reported undoubtedly saved the life of the child. Under other conditions the patient would certainly have succumbed. I also want to thank the other members of the house staff for their untiring assistance in this case.

136 West Eighty-Seventh Street.

ABSTRACT OF DISCUSSION

Dr. Willy Meyer, New York: I am sorry that I came too late to hear Dr. Goodman's paper. No doubt Dr. Goodman has mentioned how all these symptoms were worse in the course of the night, and how the house surgeon in the case of

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emergency was called on to try to save the young patient, and how, owing to his prompt action, the child's life was saved. No doubt at the time it was the proper procedure to open the trachea and to see how imminent suffocation could be averted. Certainly as to saving life it is a unique case. We do not often hear of similar cases. In the adult, however, I believe that these cases are not rare. I know that Dr. Goodman has carefully gone over the literature and collected a number of cases. I should like to report briefly one case we had not long ago under the supervision of the hospital. A young surgeon, stepping from behind a truck, walked in front of an automobile and was thrown to the ground. She was apparently not seriously injured, but an ambulance was called and she was taken to one of the nearest hospitals. On the way to the hospital the patient commenced coughing pus, and continued coughing pus, developing symptoms of lobular pneumonia, and with these conditions was transferred to our hospital with a diagnosis of some thoracic trouble, probably, on account of the expectoration of pus, tuberculosis. Expectoration of pus finally ceased, and she left the hospital after a certain time cured. We had roentgenograms made, which were of tremendous help in diagnosis. It was shown that there was peribronchial infiltration, and to all appearances—since she had had symptoms of tuberculosis before—there can be no doubt that she had a coalescence of one or more peribronchial lymph-nodes, which on account of the accident had perforated into the bronchial tract. I am sure that such cases are not rare. The patient was fortunate in that she had to expectorate the mass of pus, whereas in the interesting case brought out by Dr. Goodman there was no convenient exit for this filling up of the smaller bronchi, and if no relief had been given the child would have died.

I believe that physicians should pay more attention to these conditions, and especially insist on stereopticon examinations, which are of tremendous help in thoracic conditions.

Dr. Isaac A. Abt, Chicago: I agree with the previous speaker that this condition is not rare. It is true that Dr. Goodman himself is not positive as to the exact nature of this abscess cavity, but we have been much enlightened, especially as to tuberculosis infection, by the Vienna school, for the large number of cases examined. These reports mention almost constantly that the entrance of the tubercle bacilli is for the most part respiratory. It enters, usually, through the lung, some area in the lung constituting the initial or primary focus or lesion, similar to the initial lesion of syphilis, and soon after this there is infection of the bronchial lymph-nodes. This infection of the bronchial lymph-nodes undergoes various courses. It may form a central hyperplasia, it may remain latent or it may break into blood-vessels or lymphatics or the

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digestive tissues. The important thing is that, if we accept these views, the infection of the bronchial glands are of extremely common occurrence. Dr. Goodman's case with its brilliant treatment and unusual recovery is noteworthy, and the doctor is to be congratulated on the successful issue of the case.

Dr. A. W. Myers, Milwaukee: The paper which Dr. Goodman has presented did not mention the great help that the bronchoscope offers in these cases. It seems to me that this instrument is worth while bearing in mind in connection with the Roentgen-ray examinations, because they supplement each other so satisfactorily. A good many enlargements of bronchial lymph-nodes will, of course, occur in which no lesion can be seen with the bronchoscope, yet in many cases it is possible to obtain definite information through this means. It seems to me that the results are second only to the revelations of roentgenoscopy in aiding in diagnosing the condition.

Dr. C. F. Wahrer, Fort Madison, Iowa: Dr. Goodman says that the cases are to be divided into tuberculous and non-tuberculous. I should like to ask if they are not usually tuberculous. If not, what are they? It is well to designate distinctly all we know about this. I should like to know whether or not any but tuberculous infection is found in the mediastinum.

Dr. A. L. Goodman, New York: I agree with Dr. Abt in what he said in reference to tuberculous infection of the mediastinal lymph-nodes. I think that we all agree that, in the majority of cases, the tubercle bacillus is the exciting cause. There are cases, however, like the one which I had the privilege of presenting to you, on which we worked nearly eight months to demonstrate the existence of the tubercle bacillus, and we have failed in every instance. As I stated in my paper, we divided those cases—and from a study of the literature we are justified in doing so—into those which are tuberculous and those which are non-tuberculous. In the non-tuberculous cases, as in the one we had at the hospital, we demonstrated the presence of streptococci. You can have streptococcus or staphylococcus in any form, or a mixture of the two, and usually in the examination of a glandular exudate in which the tubercle bacillus is not demonstrated, we have a mixed infection.

The Roentgen ray shows the existence of the bronchial lymph-nodes distinctly. In these cases there is usually not much time to be lost, and to use a bronchoscope is not always easy.

There is one other point that I thought Dr. Meyer would bring out; that is, the possibility in the future, when we see large masses of lymph-nodes present by the aid of the Roentgen ray and other examinations, of determining whether or not surgical intervention would be indicated.

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REPORT OF A CASE OF ACUTE NEPHRITIS IN AN INFANT WITH CONGENITAL HEART DISEASE

H. M. McCLANAHAN, M.D.
OMAHA

History.—The infant, aged 1 year, came under my care Jan. 4, 1913. Family history negative. Parents both young and in good health. The patient had been a blue baby from birth. Breast-fed for a time. Ever since birth it had had an inspiratory grunt, more perceptible when the child was moved or held erect. Both skin and mucous surface constantly blue. The infant had steadily gained in weight, however, weighing 24 pounds at 1 year of age. Digestion had always been good, bowels regular, urine free.

History of Attack.—Dec. 5, 1912, infant was suddenly taken sick with sore throat and fever, followed by a general erythematous rash. Later this rash was followed by a series of small vesicles. December 10 some swelling about the face was first noticed. The mother said breathing became more labored and rapid, so that she had to keep the child constantly in a recumbent position. Even then she noticed that the cyanosis was deeper in color. The bowels were regular, urine becoming more and more scanty as estimated by the napkins. Infant was more stupid than usual. Jan. 4, 1913, I noted the foregoing facts as given me by the mother. Temperature 100, pulse 110, respiration 70. Child vomited two or three times a day while under my care, always after feeding. There were edema of the face, swelling of the arms and legs, and ascites, with pitting along arms and legs. Physical examination of the lungs negative. Pericardial dulness very extensive. Cardiac impulse perceptible. Blowing murmur heard everywhere over heart. Temperature did not go above 100 until a few hours before death. Respirations varied from 60 to 80 per minute.

Examination.—Urine examination January 5. Specific gravity 1.007, acid, albumin by heat and nitric acid, no sugar, no bile. Under the microscope a number of white cells, hyaline and granular casts. January 7: Examination the same with the addition of many blood-cells. Blood examination January 5: Red blood-cells, 8,140,000; white blood-

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cells, 20,000; hemoglobin, plus. Slight increase in small lymphocytes; otherwise leukocytes normal. January 7: Red blood-cells, 9,800,000; white, 16,000.

Treatment—There was nothing special in the treatment during the time that the patient was under my care. The infant responded well to hot packs. It received sodium carbonate by enema and tincture of digitalis by mouth. General anasarca decreased and quantity of urine increased. There were absolutely no nervous symptoms. Respirations continued rapid. Cyanosis extreme when infant was lifted up. Death was due to apnea.

Clinical Diagnosis.—Acute nephritis due probably to scarlet fever with congenital heart disease. I am satisfied that death was simply hastened by this disease, but was actually due to the heart lesion. In other words, the nephritis was an added burden that the heart could not carry.

Post-Mortem Made Four Hours After Death.—I desire here to thank Dr. L. Moon, intern at the Methodist Hospital, for his very thorough work in preparing the sections from various organs. The body was that of a large, well-nourished infant; slight eruption over body; chest appeared very large, no deformities, skin soft and very dark, mucous membrane dark, muscles flabby, no rigor mortis. Brain not examined. Skin of chest and abdomen quite edematous on incision. Muscles dark red and very soft. No free fluid in the abdominal cavity, no ossification in the costal cartilages. Sternum very soft and porous. Thoracic cavity contained 20 c.c. of light straw-colored fluid. No pleural adhesions. Base of heart at second rib. Apex at the sixth rib. Left border extended outside of nipple line ½ inch. Right border 1 inch to the right of the sternum. Thymus present, but no evidence of pressure on the trachea. Pericardium contained 10 c.c. of clear fluid. No adhesions. The heart was 13 cm. long, 9 cm. wide, 7 cm. thick. It will be noted that this is the size of the adult heart. Heart muscles dark reddish-brown. Cavities contained a large amount of fluid blood. It was noted that there was scarcely any clotting of blood in any organ. Veins of heart greatly dilated. Foramen ovale was open and large; indeed, there was only a light constriction between the auricles, so that both formed practically one cavity. Left auricle was greatly dilated with thin walls. The aortic and pulmonary orifices were smooth and the valves normal. The mitral valve was greatly shortened and thickened, and would not close to exceed one-half of the opening. There was also a defect in tricuspid valve. Ductus arteriosus was not found although carefully searched for. Pulmonary vessels filled with blood. It will be noted that there was practically free communication between all the chambers of the heart, no obstruction to flow of blood to lungs and that the aorta could carry a sufficient quantity to nourish the body, but that there

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was free mingling of the venous and arterial blood. The spleen was dark, congested and very vascular. Bile passages normal. Stomach dilated, also dark in color. Gall-bladder normal. Pancreas hard to touch, and dark in color. Liver 17 cm. long, 7.5 cm. thick, 12 cm. wide, dark in color, greatly congested, bleeding freely on section. Lungs dark in color but no evidence of bronchitis or pneumonia. Left kidney 7.5 cm. long, 5 cm. wide, 4 cm. thick. Right kidney 5 cm. long, 4 cm. wide, 2.5 cm. thick. Both kidneys were firm to touch and dark red on section and showed on section that they were retained by the capsule under pressure, capsule thickened. Bone-marrow of ribs and sternum very dark in color. The entire appearance was one of chronic passive congestion and the great size of the heart and liver was most striking.

Microscopic Findings.—Kidneys show a marked interstitial nephritis with chronic passive congestion. Many cells show hyaline swelling with fatty degeneration, capsule thickened. Marked round-cell infiltration in all fields.

Spleen shows marked congestion with great increase in the connective tissue.

Heart muscle is atrophic with cloudy swelling and evidence of fatty degeneration.

Pancreas shows great increase of connective tissue with marked passive congestion.

Liver capsule thickened, connective tissue increased and marked. Fatty infiltration about the center of the lobules.

Bile ducts dilated and increased pigment.

The pathologist, Dr. W. R. Lavender, reports that the sections of all organs show a passive congestion due to the congenital heart disease, with an acute process due to the complicating nephritis. This is in line with what is usually found in congenital heart disease with cyanosis.

There were two interesting features in the case:

1. Evidence of nephritis occurred earlier than is usual in scarlet fever. From a rather large experience in acute nephritis I think it is fair to assume that death would not have resulted if the infant had been normal. The absence of nervous symptoms with subsidence of the anasarca, increased excretion of urine, all pointed to a favorable termination.

2. The polycythemia. This is the second patient with congenital heart disease in whom I have found polycythemia. I have had the current literature for the last three years carefully reviewed without finding anything on the blood in infants with congenital heart disease.

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There are valuable contributions on the blood in scarlet fever and whooping-cough, but very little on the blood of infants. It seems reasonable to presume that polycythemia is physiologic in these cases; as the individual cell carries less oxygen, an increase in the number would carry relatively more oxygen. This would seem a reasonable hypothesis.

Brandeis Building.

ABSTRACT OF DISCUSSION

DR. JOHN ZAHORSKY, St. Louis: Several years ago I reported a case of polycythemia before the St. Louis Pediatric Society, in which the red cells numbered more than eight million. At that time I looked up the subject, and I am sure I found a condition described in which children with congenital heart disease showed an enormous number of red cells.

DR. C. F. WAHRER, Fort Madison, Iowa: Is any mention made in the literature that this polycythemia is compensatory?

DR. H. LOWENBURG, Philadelphia: That polycythemia is a symptom of congenital heart disease is true, but it is not pathognomonic. I recall a case in which the count was about eight or nine million and in which there was clubbing of the fingers and a murmnur. This child died rather suddenly and came to necropsy. The heart was found to be normal, but there was a marked chronic interstitial pneumonia. My diagnosis was based largely on the murmur, clubbing of the fingers, dyspnea and polycythemia. I was wrong. The physical signs of interstitial pneumonia were lacking.

DR. H. M. MCCLANAHAN, Omaha: I have looked in the archives for ten years and have found no literature on this subject. All patients with congenital heart disease whom I have ever seen ultimately die of some intercurrent affection. I have had one live to be 16, the oldest patient of whom I have any knowledge. These patients are especially prone to acute infections, and, if they have even a mild infection, it is most likely to prove fatal.

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ACUTE ACID INTOXICATION IN CHILDREN

THOMAS C. McCLEAVE, M.D.
BERKELEY, CAL.

This paper contains nothing new; but because the subject receives scant discussion in current text-books, and periodical literature contains articles dealing rather with particular aspects of the matter, it may be of some value to make a general survey of our knowledge of that important disorder occurring in children which, for want of a better term for the time being, we may still tentatively designate "acute acid intoxication."

Acid intoxication is an extremely common phenomenon in a variety of pathologic conditions, but in children, usually from 2 to 10 years old, we see very frequently a symptom-complex in which acidosis is a marked and constant characteristic. Attacks are commonly recurrent, and may come on suddenly or be preceded for a day or two by a prodromal period in which are observed listlessness, anorexia and coated tongue, and, quite strikingly, a bluish pallid ring around the mouth and beneath the eyes, with dusky cheeks. Headache may be complained of, and there is usually a coryza and injection of the conjunctivae, with a variable cough. Intense nervousness, even to marked meningismus, may be exhibited in the beginning, though always as the attack progresses, and frequently throughout, the child is apathetic and, in extreme cases, may even develop coma. In any but mild cases, the appearance of the child is indicative of serious illness. The face is pinched and pale, or often cyanotic, the eyes, at first bright, become sunken and dull, the lips are dry and cracked, the teeth covered with

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sordes, and the tongue dry and brown over the dorsum, with "strawberry" edges. The abdomen is retracted, and general wasting may be extreme. The skin is dry and harsh, often scaling after a severe attack.

The breath is foul, but the sweetish, fruity odor of acetone can always be detected, and frequently permeates the entire room in which the patient lies. There is always fever, the temperature rarely exceeding 102 or 103 F., but occasionally being very high. The pulse is soft, rapid and irregular, and the respirations sighing and irregular, often slow and deep, with marked "air hunger," both clinical and experimental evidence seeming to show that the acid poisons have a direct depressing effect on the vasomotor and respiratory centers.[1] I have seen two cases in which asthmatic manifestations were a prominent feature of the attacks.

There is no desire for food, and vomiting, except in the mildest cases, is always present, and often is extremely intractable. Large amounts of fluid may be vomited, and the rapid dehydration of the tissues occasions a most intense thirst, the child constantly pleading most piteously for water. The bowels may be constipated, or, less frequently, loose, with green or gray foul stools. The urine is scanty, and may contain albumin, casts and blood. In one case under my observation the child passed only a few ounces of dark-green, syrupy urine in twenty-four hours, which solidified on boiling.

Prostration, in severe cases, is always marked, and not infrequently a fatal termination seems imminent; but deaths from this cause are very rare. The duration of the attacks is from one or two to seven or eight days.

Diagnosis, when one is familiar with the condition, is usually not difficult. Acute digestive disorders may be excluded by the absence of a history of dietetic errors, and the persistence of the vomiting and other symptoms; acute appendicitis by the absence of pain, tenderness, and rigidity; disease of the brain or kidneys, or begin-

1. Ehrman: Berl. klin. Wchnschr., 1913, I, 49.

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ning meningitis, at first readily suspected, by the progress of the attack, and the early development of the characteristic urinary findings. That acidosis and vomiting are frequently observed at the onset of an acute infectious disease may sometimes mislead one, and seeing a child for the first time with an attack of the kind we are considering, I am in the habit of mentioning this to the parents, lest pneumonia or one of the exanthems unexpectedly supervene. A history of similar previous attacks of course aids in determining the nature of the present one.

The etiology and pathology of this condition are still obscure. Because of the infrequency of death from this cause, we have almost no necropsy data. Necrotic changes in the gastric and intestinal mucosa,[2] degeneration of the glandular epithlelium, small hemorrhages and ulcerations, fatty infiltration of the liver,[3] and in one case, hypertrophic pyloric stenosis,[4] are about all that have been observed.

Marfan[5] in 1901 noted the presence of acetone with recurrent vomiting, and Edsall[6] in 1903 directed attention to the occurrence of large amounts of acetone and diacetic and oxybutyric acids in the urine. Since that time discussion of the subject has largely centered about the question of the rôle of these bodies in the production of the symptoms observed, whether they are of etiologic importance or merely morbid products of the underlying pathologic process.

Langmead[7] in 1906 doubted that they were the cause of the vomiting and other symptoms, stating that he never found them in the urine until well after the onset of the attack. Two years later he ascribed the production of the acid poisons to deficient oxidation of fats by the liver consequent on impairment of its functions by

2. Griffiths: Am. Jour. Med. Sc., 1900, cxx, 553.
3. Langmead: Brit. Med. Jour., 1907, ii, 819.
4. Russell: Brit. Jour. Child. Dis., 1910, vii, 49.
5. Marfan: Arch. de med. des enf., 1901, iv, 641.
6. Edsall: Am. Jour. Med. Sc., 1903, cxxv, 629.
7. Langmead: Brit. Med. Jour., 1905, i, 350.

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toxins derived from the gastro-intestinal tract, or bacterial activity in other parts of the body.[3]

Morse[8] considered the acid bodies a secondary manifestation, but believed that they were, in part at least, responsible for many of the peculiar symptoms.

Ratchford[9] considered the condition one of auto-intoxication, functional hepatic derangement causing an accumulation of purin bodies in the blood.

Ewing[10] regarded the trouble as a disturbance of fat metabolism, due to defective activity of the liver, associated with absorption of poisonous putrefactive products from the intestine.

Chronic appendicitis was assigned an important etiologic significance by Comby.[11]

Ely[12] asserted that the disorder was a neurosis, and Howland and Richards[13] ascribed it to an unstable state of the nervous system and regarded shock or excitement as the cause of a disturbance of metabolism leading to diminished oxidation, with the circulation of the unoxidized substance in the blood. Such substances being excreted into the stomach, the vomiting may be considered as an attempt of the body to rid itself of them. My own experience with these cases indicates the undoubted importance of the nervous element in their etiology. I see very many of them, for example, at such times as Christmas week, and following children's parties, in little patients in whom the question of dietetic errors as a factor may be entirely excluded.

Janeway and Mosenthal[14] thought that carbohydrate starvation might be a causative factor, as a deficiency of carbohydrates in the diet will produce an acetonuria.

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8. Morse: Arch. Pediat., 1905, xxii, 571.
9. Ratchford: Arch. Pediat., 1904, xxi, 881.
10. Ewing: Cornell Univ. Pub. Dept. Pathol., 1908, viii.
11. Comby: Arch. de méd. d. enf., 1905, vii, 741.
12. Ely, Thomas C.: Cyclic Vomiting in Children, THE JOURNAL A. M. A., March 28, 1903, p. 845.
13. Howland and Richards: Arch. Pediat., 1907, xxiv, 401.
14. Janeway, Theodore C., and Mosenthal, H. O.: An Unusual Paroxysmal Syndrome, Probably Allied to Recurrent Vomiting with a Study of the Nitrogen Metabolism, Arch. Int. Med., October, 1908, p. 214.

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Sedgwick,[15] in 1910, called attention to the occurrence of creatin in the urine of a child subject to recurrent vomiting, and later,[16] reported further cases in which creatinuria was observed. Mellanby[17] reported similar findings in the case of a boy aged 6, in whose urine creatin was always present, increasing in amount before the onset, and reaching a high level with the climax of each attack, the condition of the patient as regards acidosis apparently being immaterial, as the acid bodies usually considered at fault were sometimes present in large amount without causing any apparent change in the child's well-being. The attacks were considered as due to intestinal toxemia, with possible infection of the portal area, the acidosis being merely secondary to derangement of the glycogenic function of the liver, which, causing imperfect catabolism of the fats, an accumulation of aceto-acetic and beta-oxybutyric acids in the body results, these substances being intermediate products in the breaking down of the fats, which normally should proceed to the formation of carbon dioxid and water. The presence of creatin is explained by the fact, as shown by Cathcart,[18] that a deficiency of carbohydrates, or inability to utilize such as are present in the body, will cause this substance to appear in the urine in large amounts. The hypothesis was advanced by him that carbohydrates are essential for protein metabolism, as they are for fat metabolism.

Finally, acidosis is common in children exhibiting catarrhal conditions of the nasopharynx, adenoids and diseased tonsils, and as before noted, acute coryza is a marked premonitory symptom of the attacks. Griffiths,[2] Comby,[11] Eustace Smith,[19] Sedgwick[20] and others have

15. Sedgwick, J. P.: Creatinin and Creatin Metabolism in Children, THE JOURNAL A. M. A., Oct. 1, 1910, p. 1178.
16. Sedgwick, J. P.: Studies of Recurrent or Periodical Vomiting, Am. Jour. Dis. Child., April, 1912, p. 209.
17. Mellanby: Lancet, London, 1911, ii, 8.
18. Cathcart: Jour. Physiol., 1909, xxxix, 311.
19. Smith, Eustace: Lancet, London, 1911, ii, 1186.
20. Sedgwick, J. P.: Studies of Recurrent or Periodical Vomiting, Am. Jour. Dis. Child., April, 1912, p. 212.

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emphasized this relationship, and have noted marked improvement in these cases after removal of adenoids and tonsils, or other treatment of the nose and throat. I am convinced from my own observation that there is no other single factor so important, as a large proportion of the children I see with acidosis have bad throats and the recurrent attacks of acidosis and vomiting are relieved by proper treatment of the throat conditions.

Summing up and correlating all these various views as to the etiology of the condition, I may say that in children who suffer from periodic attacks of acid intoxication or recurrent vomiting, there is probably almost always some chronic focus of infection, adenoids, diseased tonsils, an inflamed bowel or appendix, or what not, the toxins from which, being constantly absorbed, act primarily on the liver. From time to time, by reason of the cumulative effect of the toxins, or to further disturbance of the already impaired equilibrium of metabolism by dietetic indiscretions, or by excitement, fatigue or other nervous element, there results a failure of the liver properly to perform its functions. The processes of carbohydrate metabolism are first disarranged, the frequency and severity of the attacks in childhood, perhaps, being due to the fact that the reserve supply of glycogen in the liver is not so great, proportionately, in children as in adults, and that therefore any interference with the glycogen-storing or glycolytic powers of the liver, there being no longer a proper supply of sugar available, induces an immediate disturbance of fat and protein metabolism, manifested by the appearance in the urine of the toxic bodies before described. How these operate to produce the prostration, vomiting, rapid wasting, vasomotor changes, nervous disturbances and other characteristic symptoms cannot yet be explained.

The consideration of treatment is largely omitted from the literature, or often discussed with a note of pessimism which is entirely unjustified to-day, as the little patients who suffer from this disorder offer excellent

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opportunity for successful therapy. Of fundamental importance, of course, is the determination and proper treatment of such foci of infection as have been referred to. Adenoids and bad tonsils will, I believe, be most commonly found, and their removal is usually followed by complete cessation of the acidosis attacks. Similar relief has frequently resulted from appendectomy in properly selected cases. Next in importance may be placed a properly balanced dietary, low in fats, with ample amounts of carbohydrate in the form of suitable breadstuffs, crackers, dry cereal preparations, rice, etc., with simple sweets and milk and lean meats in moderation, with plenty of green vegetables and fresh fruits. Citrus fruit juices are of especial advantage because of their content of potassium citrate which is converted in the body to potassium carbonate, and so tends to maintain the alkalinity of the tissues. Grape-juices, being rich in alkaline salts, are also valuable.

Constipation may be overcome by dietetic measures or by mild saline aperients if necessary, and the child's general hygiene should be carefully conserved by a quiet outdoor life with avoidance of too great fatigue and excitement. Drugging is not to be recommended except that in children with frequent recurrence of attacks, a periodic thorough emptying of the large bowel with a saline laxative or castor oil, with the administration of a dram or two of sodium bicarbonate one day per week may be beneficial. The routine daily use of an alkali is, of course, inadvisable because of its deleterious effect on gastric digestion.

Impending attacks, if recognized, may often be aborted by prompt catharsis and the free use of sodium bicarbonate as recommended by Edsall,[6] and favorably reported on by Pierson[21] and many others. Routine examinations of the urine will often give warning of the approach of an attack even before the onset of prodromal symptoms. In the treatment of an established attack,

21. Pierson: Arch. Pediat., 1913, xx, 505.

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the bicarbonate is valuable not because of any neutralizing effect on the acid bodies, but because it stimulates their excretion, and probably exerts a protective influence on the body fats. It may be given by stomach, if vomiting does not prevent, or preferably by rectum, in doses of 60 grains in 3 or 4 ounces of water every three or four hours, or more or less continuously by the drip method; and in extreme cases, by intravenous injection. To meet the need for carbohydrate, sugar must be given in that form in which it is most available for utilization, namely, dextrose. It, too, may be administered by mouth, by rectum, in 4 per cent. solution with alkali, or by hypodermoclysis if the symptoms are urgent. So-called "corn-syrup," sometimes recommended for this purpose, is commercial glucose, derived from starch by the action of hydrochloric acid under pressure of steam, and as it contains only about 30 per cent. of dextrose and over 50 per cent. of dextrin, it should not be used if true glucose can be obtained.

Extreme drying out of the tissues may be counteracted by saline infusions, prostration by appropriate stimulants, and nervous symptoms by opium, chloral or bromids, or by ice-packs or moist packs. As the vomiting subsides, gruels may first be given with orangeade or other fruit-juice drinks, gradually increasing the diet to normal.

Convalescence is frequently surprisingly rapid, and the little patient, except for the wasting, will soon show no signs of what, a few days previously, appeared to be so serious an illness.

Berkeley National Bank Building.

ABSTRACT OF DISCUSSION

Dr. John Zahorsky, St. Louis: Dr. McCleave describes the color of the cheeks as being dusky. One of the most important symptoms of acid intoxication is a deep flushing of the cheeks, but I would not call it dusky. It is really a scarlet. The diagnosis is almost certain at a glance when the child has this peculiar color and is breathing very rapidly. I recall the case of a child who had these attacks and who,

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after circumcision, had no attacks for nearly a year. Of course, the mother attributed the improvement to the operation. The operative shock does seem to modify certain phenomena. The removal of the appendix, for instance, no doubt has a beneficial influence on nervous diseases.

I believe that this disease is related to migraine. I recall a family in which the mother has severe migraine attacks. One child has asthma, another has eczema and another has cyclic vomiting, showing a nervous disturbance throughout the whole family. I have noticed marked enlargement of the liver in two severe cases. The liver should be palpated in all cases. It has been definitely established that enlargement of the liver does occur in these severe cases. One patient was very sick. We thought the child would die. I marked out the liver on the abdomen daily and found it extending several inches below the margin of the ribs. On a subsequent visit I detected that it had receded half an inch. I then gave a favorable prognosis, although the other symptoms were still as severe as before. I would give enlargement of the liver as an additional symptom to be watched for in severe cases of this disease.

Dr. J. R. Snyder, Birmingham, Ala.: Dr. McCleave has not mentioned a certain type of case common in the South. It occurs in children younger than those indicated by the age limits he gave; most commonly in infants between 8 and 18 months of age. It is characterized by an acute onset, vomiting, increasing dyspnea and enlargement of the liver. In the article read by Dr. Thomas D. Parke in this Section several years ago, the symptom-complex of the disease was said to consist of a low-grade temperature, as a rule, intense thirst, vomiting, increasing dyspnea, constipation, dark green or tarry stools, enlargement of the liver, such urinary findings as occur in any acidosis, and so on. We see a large number of these cases in the South. We have come to regard them as among the most serious with which we have to deal. The mortality is over 80 per cent.

Dr. C. H. Johnson, Grand Rapids, Mich.: This is a comparatively new disease. About eight years ago I had a severe case. The boy had been ill ten days with recurrent vomiting and was nearly dead when I first saw him. These children have attacks of indigestion and vomiting, and we are apt to think of a gastro-intestinal trouble. We simply do not recognize the condition. My own son had recurrent vomiting for four or five years before I made a correct diagnosis. These attacks were never ameliorated in the least until I had thoroughly removed the stumps of tonsils that had been taken out several years before that. With complete removal of the tonsils he recovered. The case I referred to before was the worst I ever saw. The attack always began

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with stuffiness in the nose and a slight sore throat, and what I concluded was inflammation of very small adenoids. That boy did not make any improvement until the nose and pharyngeal space were put in good condition.

Dr. Thomas B. Cooley, Detroit: I am glad that Dr. Snyder spoke of the severe cases in babies. I think that they deserve to be distinguished from the type of acidosis which we find in so-called recurrent vomiting. I have seen a number of these cases in babies anywhere from 7 to 18 months or 2 years of age. The acidosis seems to be an accompaniment of an acute infection—often of the influenza type. Prostration is great from the start. The children do not always have a low temperature—I have seen it 103 F. and over—and they have great air hunger, severe prostration, not much vomiting, perhaps, and not so severe or so difficult to check as in cases of recurrent vomiting in which there is a strong nervous element.

Dr. I. A. Abt, Chicago: It seems to me that the altered constitutional state due to abnormal acid in the urine must be somewhat more carefully differentiated than has been the custom in the past. Acetone, diacetic acid, butyric acid, creatin and creatinin may all be recovered from the urine of children ill with various conditions. Acetone and diacetic acid occur in normal children. Undoubtedly they also occur in various forms of infection. The class of cases that I wish to refer to are those considered by Dr. Parke in this Section some years ago and referred to this morning by Dr. Snyder. I have seen a number of these cases. I am sure that the condition is not so infrequent as would be supposed from the scant reference in the literature. There is a group of babies, from 8 to 18 months of age, particularly at the weaning period, who fall ill with gastro-intestinal symptoms consisting of vomiting, slight diarrhea and rapid pulse, with a low temperature. The first striking symptom is the rapid breathing. Some pallor is noticed and, by and by, great prostration; soon constipation occurs, followed nearly always by more or less complete loss of intestinal tone, with symptoms which may simulate an obstruction. The baby passes neither gas nor stool. I am sure that this is a definite type of case. I have seen a number of them, some even during the time when breast-feeding was the exclusive diet and some during the transitional period from breast-feeding to that of artificial feeding. The urine contains acetone, diacetic acid and sometimes leucin and tyrosin with a trace of albumin. A brief reference to some case histories may throw light on the course and etiology of the disease.

A healthy baby was born to parents in perfect health and remained well until about the ninth month, when it was weaned. In a few days he developed the symptoms of gastro-

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intestinal disturbance mentioned previously and died in a few days. Some time later another baby was born. He also was healthy and strong and had never been sick up to the ninth or tenth month. He developed the same symptoms and died. The urine contained acetone, diacetic acid, oxybutyric acid, creatin and creatinin.

Another baby was born. The same symptoms developed. We examined the mother's milk and urine carefully. The breast-milk showed nothing, but the urine contained a small amount of sugar. One night the baby seemed about to die. We gave large doses of carbonate of soda, took away fat and gave skimmed milk, soy-bean flour, gelatin by mouth and glucose solution per rectum. We felt that starvation was possibly an important etiologic factor in producing these acid conditions. Eight per cent glucose solution with normal salt solution we believed might be absorbed without being oxidized in the liver, and possibly counteract the intoxication. A 2 per cent. solution of sodium carbonate was given under the skin. The baby was better the next day and recovered in a few days. The carbonate of soda solution produced an immense slough, as large as the palms of two hands.

What caused this intoxication? We have concluded from experiment that it was not the breast-milk. The mother's urine cleared up in a few days and the sugar disappeared. We thought the fact that two other children had been similarly ill pointed, first, to a family type of disorder, and secondly, to something wrong in the metabolic functions of this baby; that some defect of the organism rendered it impossible for him to take care of some particular foodstuff. Was it a fat intoxication? It did not present the symptoms or reactions usually observed in fat intoxication of infants. Nor did the condition correspond to any known clinical type of sugar intoxication. Possibly this severe and unusual form of acid intoxication is due to some albumin, the nature of which we are unable to determine at present.

Dr. J. L. Morse, Boston: I have written and talked a good deal about acid intoxication. I thoroughly believe that there is such a condition in children, because I have had cases in which parents recognized the odor of acetone bodies in the breath and urine and in which they have been found present in large quantities before the onset of symptoms. I believe that it is the cause of some cases of recurrent vomiting. Someone said that the condition was not being recognized as often as it should be. My experience in the last year or two has been, not that it is not recognized, but that a great many other things are being called acid intoxication that have no relation to it, the reason being that someone has found a little acetone or diacetic acid in the urine. Anyone who has examined a large number of urines will be surprised

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to find how often these substances are found in the urine and how infrequently there are any symptoms from them. There are symptoms only when it is present in a considerable amount. One must be extremely careful in making diagnoses of said intoxication in children with vomiting and other symptoms simply because a small amount of these substances are present in the urine.

Dr. H. T. Price, Pittsburgh, Pa.: One fact that has impressed me is the tendency to consider this condition as being of nervous origin. So serious a condition is hardly of nervous origin, because we know from experience that if we permit a child to live properly we can prevent many of these attacks. Ordinarily we believe that acidosis is a serious condition, but that most patients recover. I am glad to learn that others have given a grave prognosis, because some patients have died under my care, and I have felt that I was at fault for not getting the results that we are led to believe should be obtained.

Dr. L. R DeBuys, New Orleans: I concur in the remarks of Dr. Abt, and I also feel that Dr. Morse has sounded a strong note. There is the other side to be considered, however. The tendency is for physicians to ridicule acid intoxication, and the sooner they appreciate the gravity of the condition the sooner more lives will be saved.

Dr. Zahorsky has made the same observation that I have regarding the peculiar flushing of the face. The flushing, at times resembling that of scarlet fever, is suggestive. As these patients improve, I have noticed the peculiar flushing disappear.

Another symptom of value in the diagnosis is that the temperature is usually low. The condition may start with some digestive disturbance and a somewhat high temperature. After the bowel has been cleaned out the temperature will decline. In fatal cases there is usually a gradual rise of temperature, signifying, I believe, that the means of eliminating poisons are locked up. These patients die, as a rule, with hyperpyrexia.

Dr. H. Lowenburg, Philadelphia: The point Dr. Morse brought out is important and calls to mind the history of two cases that came under my experience. One illustrates that not all cases of recurrent vomiting in children are due to acidosis. The child was 4 years of age. A diagnosis of acidosis had been made. On careful investigation of the urine no evidence of acidosis was discovered. Roentgenoscopy indicated the presence of a marked degree of pyloric spasm. An interesting feature was that the child recovered after half a dozen stomach washings and has had no recurrence for about five months.

The other was the case of an infant sent to me for recurrent vomiting with the diagnosis of pyloric obstruction. Here,

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too, a roentgenogram was made, and it showed kinking of the bowel in the region of the sigmoid. The infant was much constipated. The kink was the only thing that could be discovered. The pylorus was clear. The vomiting was probably reflex or toxic. This infant, too, recovered with use of high enemas of olive-oil.

Dr. Abt referred to a slough that followed the subcutaneous injection of bicarbonate of soda solution. I have given bicarbonate of soda by hypodermoclysis in about seven cases, and in each there was a slough.

Dr. Thomas C. McCleave, Berkeley, Cal.: With reference to the comment of Dr. Zahorsky and Dr. DeBuys in regard to the flushing of the face, I used the term dusky as best describing the appearance at the height of the attack. It is characteristic in the beginning that these children have a bluish, pallid ring under the eyes, sometimes a very intense white ring around the mouth and flushed cheeks. As the attack progresses and the intoxication becomes more intense, air hunger and cyanosis develop, diacetic and oxybutyric acids having a direct depressing effect on the respiratory and vasomotor centers.

I think that adenoids and diseased appendixes are of importance as sources of toxemia. We know that the liver is one of the most susceptible organs in the body to toxins of any nature. A disturbance of liver function results, and, unquestionably in my experience, removal of adenoids and bad tonsils or appendixes has worked a marvelous change sometimes in a child subject to attacks of this nature.

I purposely omitted from consideration the attacks in younger children referred to by other speakers as having been first described by Dr. Parke, as I am not at all sure that they belong in the category of the cases I had in mind.

Great care is necessary in the use of either sodium carbonate or bicarbonate under the skin or intravenously, as ordinary preparations of these drugs are contaminated; it is exceedingly important to have a chemically pure drug, or sloughs will occur. Dr. Fischer maintains that sodium carbonate is more valuable for the relief of acid conditions than the bicarbonate, for the reason that the sodium in the latter has already tied up to it as much acid as it will carry, and sodium carbonate has therefore a better combining value.

Dr. H. Lowenburg, Philadelphia: The last case of sloughing I had followed the use of Fischer's solution.

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THE PROTEIN POISON AND ITS RELATION TO DISEASE

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ANN ARBOR, MICH.

In 1903, Vaughan and Wheeler found that the cellular substance of the colon bacillus contains a highly active poison. This was obtained by extracting the dried, dead cells with a 2 per cent. solution of sodium hydroxid in absolute alcohol at 78 C. (172.4 F.). This poison was partially purified by repeated precipitation from alcoholic solution with either mercuric or cupric chlorid and removal of the base with hydrogen sulphid. This poison kills guinea-pigs when injected intravenously in doses of 0.5 mg. When given intra-abdominally this dose must be multiplied by 16 and when given subcutaneously by 32 in order to cause death.

Subsequently the same, or a closely related, poison was obtained from other pathogenic and from non-pathogenic bacteria, and then from vegetable and animal proteins. This work has been repeated and confirmed by others in this country and abroad. It is now known as "the protein poison" and is believed to be a constituent of every true protein whatever be its source. The purest proteins known, such as edestin from hemp-seed and zein from corn-meal yield this poison. It is not found in certain albuminoid substances, such as gelatin. It is found in Witte's and De Chapateau's peptones, but not in that of Defresne. Whether or not the latter is prepared from gelatin is not known.

The symptoms induced by this poison are quite characteristic and can be divided into three well-defined

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stages. The first is that of peripheral irritation and is marked in the guinea-pig by attempts to scratch every part of the body. In man it is shown by an urticarial rash which spreads from the point of injection and may extend to every part of the skin and invade the visible mucous surfaces. The second stage is one of partial paralysis with rapid and shallow breathing. The third stage is known as the convulsive, and begins in isolated clonic movements which become more frequent and general, finally involving all the muscles of the body and terminating, as a rule, in death. With fatal or nearly fatal doses there is a fall in temperature, which with fatal doses is progressive, and the temperature may fall in guinea-pigs to 85 F. before death. When recovery occurs, the first symptom of this is a return of the temperature toward the normal. In both man and experimental animals, recovery from a single non-fatal dose is speedy, and a complete return to the normal condition may result within a few hours.

Small doses of this poison administered subcutaneously cause fever, and a like effect can be induced with unbroken proteins. By changing the size of the dose and the intervals of administration it is possible to induce any and every known type of fever in experimental animals. One can induce an acute fever, terminating fatally, in a few hours. In this type the temperature can be carried to 107 F., which seems to be near the thermal death-point in these animals. Intermittent and remittent fevers are easily established and varied at the will of the experimenter. A prolonged fever extending through weeks and with a chart not distinguishable from one of typhoid may be established and carried to a fatal issue in rabbits. In fever of this kind there is the usual morning recession and the afternoon exacerbation so characteristic of typhoid. Furthermore, there is increased nitrogen elimination, and progressive emaciation is observed.

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When proteins are taken into the alimentary canal and are acted on by the digestive juices the product becomes poisonous at about the peptone stage, and if the peptone formed in alimentary digestion should be absorbed into the circulation it would be highly injurious; but the digestive process proceeds and the peptone is broken up into harmless amino-acids, which are absorbed and synthesized into the proteins of our bodies.

When proteins find their way into the blood without being brought under the action of the digestive juices of the alimentary canal they must be digested in the blood and tissues, and in this process the protein poison is set free and, since general distribution cannot be hindered, it exerts its deleterious effects on the body.

We now know that the symptoms of many diseases are due to the parenteral digestion of proteins. Whenever a foreign protein, be it particulate or in solution, alive or dead, gets into the blood and tissue it must be disposed of by parenteral digestion. There seems to be no great differences in the amount of poison contained in the molecules of ordinary proteins, whether they be vegetable or animal, and, as has been said, the action of the poison is the same whatever the protein from which it has come. Dead protein contains just as much poison as living protein, but if the latter can grow and multiply in the body it increases the amount of its poison. Quite naturally most of the foreign proteins that find their way into the blood are living, such as bacteria and protozoa. Researches into the poisonous action of ordinary proteins, however, under the influence of the condition of sensitization, have demonstrated that living proteins are not absolutely essential to the development of certain diseases. If in any way proteins find access to the blood and tissues and the body becomes sensitized, then disease and death may result without bacteria or protozoa playing any part in the process. It has been shown quite conclusively that hay-fever and kindred disorders are due to sensitization to the pollen of diverse

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plants. Horse-asthma comes from the inhalation of hairs, and possibly of other proteins, from the horse carried through the air in other forms. The inhalation of peptone or other finely divided proteins may cause acute and subacute inflammation of the upper air-passages. Sensitization may result from the absorption of undigested or partly digested proteins from the alimentary tract. Whether or not the summer diarrheas of infancy ever originate in this way is a question to which a positive answer cannot yet be given. There are some good reasons for suspecting that the lowered vitality of the infant by excessive heat may lead to absorption of undigested proteins, and in this way cholera infantum and kindred diseases may be induced. We have not enough information on this matter at present, however, to enable us to speak positively, and I shall confine myself to a brief statement of the part played by the protein poison in the bacterial diseases. Recent studies in protein sensitization have quite materially modified our views concerning the nature and progress of the infectious diseases.

Bacteria have generally been regarded as unicellular plant organisms. This view is without just warrant. There is no proof that the bacterial cell contains cellulose. Indeed, all the evidence that we have concerning the chemistry of the bacterial cell is quite to the contrary. The bacteria which have been most thoroughly studied consist largely, if not wholly, of nucleoproteins or glyconucleoproteins, and are therefore more closely related to animal than to vegetable forms of life.

It has been assumed, furthermore, that the chemical structure of the bacterial cell is relatively simple. This has been shown not to be true. The molecules which make up the greater part of the proteins of the bacterial cell are quite as complicated as those that are found in the cells of our own bodies. These molecules contain at least two carbohydrates, one of which is attached to the nuclein group, while the position of the other has not

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been determined; but the weight of evidence is that it is a constituent of the protein part. Bacterial cell-substance yields the nuclein bases, and the greater part of it consists of protein, as is shown by its abundant yield of amino-acids, both mono and diamino. It will be seen from this that, although bacteria may be morphologically simple and without marked differentiation in structure, they are chemically quite complex and highly organized. This means that functionally they are quite on a par with the cells of the animal body with which the pathogenic bacteria so often compete.

The newer theory of how bacteria cause disease may be stated as follows: The cell is the morphologic unit of life, though not the physiologic unit. The latter is the protein molecule which lies in the cell and of which the cell is essentially composed. Bacteria are particulate proteins, and viruses capable of causing disease may be without form recognizable by us. The only essential and constant distinction between living and dead matter is that the former is never in a state of equilibrium; it is constantly absorbing and excreting; it feeds and eliminates; it is constantly trading in energy; it is labile, not stable. Every living cell must form ferments by which it splits up the pabulum on which it feeds. Whether a given bacterium is pathogenic to a given animal or not depends on two things. First, in order to be pathogenic it must be able to split up and feed on the proteins of the animal body; otherwise it cannot grow and multiply in that animal's body and consequently cannot harm it. Secondly, the ferments of the cells of the animal's body must not be immediately, at least, destructive to the invading bacterium. When the ferments of the body-cells have this destructive action on the bacterium, the latter cannot be harmful to the former. These two things determine the pathogenicity or non-pathogenicity of a bacterium, and one or the other or both lie at the base of all bacterial susceptibility and immunity. The pathogenicity of a bacterium is not determined by its

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capability of forming a poison, because there is no protein without poisonous content. The ferments with which cells, be they bacterial or body cells, split up their pabulum or prepare their food are known as extracellular ferments. They diffuse from the cell into the surrounding medium, and exert a cleavage action on those food substances which they are capable of thus altering. Some are highly diffusible and may pass through a relatively large amount of pabulum, while others apparently act only on those substances that come into contact with the cell.

Of all the bacteria with which we have worked the *Bacillus prodigiosus* yields the largest amount of the protein poison, but this organism is not pathogenic to man or laboratory animals because it cannot grow and multiply in the animal body. On the other hand, the anthrax bacillus yields a relatively small amount of the protein poison, and yet it is one of the most infectious organisms because it can grow and multiply in the animal body.

The extracellular ferments are not the only ferments with which living cells are supplied. Many, probably all, living cells have intracellular ferments, which are non-diffusible and remain within the cell. The function of these is to take the food as prepared by the extracellular ferments and build it into the cell tissue. It will be seen that the extracellular ferment is destructive or analytic in its processes, while the intracellular ferment is constructive or synthetic.

The important lesson which we have learned from our studies of protein sensitization is that the nature and action of cell ferments may be, to some extent at least, modified at will. Cells can be trained or educated to pour out a special ferment to digest a specific body. This is the explanation of vaccination. The virus of small-pox is pathogenic to the unvaccinated, but is non-pathogenic to the man who has had the disease or has been vaccinated. The typhoid bacillus is no longer

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infectious to the man who has been properly vaccinated against this disease. Vaccination is secured by introducing into the body that protein which causes the disease, but in a condition so modified that it will no longer cause the disease, but will lead the body-cells to pour out a ferment which will destroy that specific protein when it is reintroduced. In vaccination for small-pox the protein has been modified by being passed through the cow. A vaccine for anthrax is obtained by growing the bacillus at a high temperature or in the presence of a weak germicide. The dead typhoid bacillus serves as a vaccine. When an individual has been protected by one of these vaccines and is exposed to the disease, the organism is destroyed so soon after admission to the body that it does no harm, and in this way immunity is secured by vaccination. It is easily understood that this form of immunity is not absolute, but is relative, and may be overcome by severe or prolonged exposure.

Vaccines are now used not only as preventive but also as curative agents. Much harm is being done by their indiscriminate and ignorant employment, but their proper use is rational and scientific. They are of special value in local infections, and they operate by stimulating a wide area of body-cells to furnish a ferment which destroys the bacterium that is the cause of the local condition.

In the light of what has been learned from studies of the protein poison and protein sensitization let us see how the typhoid bacillus acts in inducing the disease. The bacillus finding its way into the body grows and multiplies. Its extracellular ferment acts on the proteins of man's body and prepares the food which is built into typhoid tissue by the intracellular ferments. The ferments of the body-cells are not capable of destroying the invading organism and during the period of incubation it multiplies greatly. During this time the man is not ill. It is not, therefore, the growth and multiplication of the bacillus in the man's body that

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directly causes the symptoms of typhoid fever, because at the time of its most active growth there is no fever or other symptom. During the period of incubation the bacillus furnishes the ferment, and the proteins of the man's body constitute the substrate. These proteins without much change are taken into the bacterial cells and built into typhoid bacterial tissue. The process is largely one of assimilation and construction, and no poison is liberated; consequently there is no fever or other symptom. After about ten days the period of incubation ceases and the disease is ushered in. The body-cells, under the influence of the foreign protein and for the purpose of its destruction, begin to pour out a new specific ferment. During the disease the body-cells furnish the ferment, and bacterial cells constitute the substrate, the process is destructive, the protein poison is liberated and fever with its concomitants result. The person may die as a result of the too rapid liberation of the poison, from a lesion due to the effects of the products of the parenteral digestion or from chronic poisoning.

221 South State Street.

ABSTRACT OF DISCUSSION

DR. JOHN ZAHORSKY, St. Louis: It is refreshing to pediatrists to find that many American pediatrists and physicians have rather held to the original view that, after all, it is the protein that causes the death of the babies in summer diarrheas. Holt of New York has held the same view. There are certain forms of diarrhea in children that are not bacterial. I think that this is now generally recognized. The method by which this poison is produced, how it enters the blood and what circumstances produce it, is yet to be worked out. These diarrheas occur early in the summer. The condition is ordinarily called fermentative diarrhea. It is attributed to carbohydrate fermentation. While we have acute attacks of indigestion in the winter, due to acute indigestion of sugar, in the summer we have diarrheas which we cannot cure by giving the various treatments. The diarrhea is persistent, sometimes for weeks; that type of disease, the toxin of which is unknown, is probably not a direct infection, but undoubtedly depends on some poison, as suggested by Dr. Vaughan.

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Dr. Mary Strong, Omaha: I want to speak from the clinical point of view. In Omaha the climate is abominable in summer. Some ten years ago a healthy, live baby was born under my service. I saw that baby when it was about three days old, and for no reason that we could discover it was almost comatose, with a temperature of over 104 F. I did not have laboratory methods convenient for use. I used common sense. I had the baby stripped and sponged with cold water; cold water was injected into its rectum, and we gave it all the cold water that it would swallow; the baby promptly recovered without any change of food. It was having mother's milk, which was good. I know now why that baby recovered. Since then, every summer I have seen babies in Omaha dressed according to the directions of the Eastern doctors, with flannel next to the skin. My first treatment is to take off every scrap of flannel. I think that the nearer naked the baby is in hot weather the better. I saw a baby in my office last summer with a high fever and acute diarrhea, dressed according to Holt. It was not dressed that way when it left my office. It had on one necessary garment and a slip—and I told the mother to take that off when she reached home. The baby recovered. I believe that I know now why it recovered. I found out several years ago that clothes kill a great many babies in the summer time, particularly flannel. It makes them so warm that changes take place in the digestive juices.

Dr. C. H. Johnson, Grand Rapids, Mich.: Finkelstein has reduced the mortality in his institution from 60 or 80 to 7 per cent. by cutting out sugar and salt in a large degree. He agrees with everything else that Dr. Vaughan says. Finkelstein says that the death-rate in diarrhea has not been reduced by the sterilization of milk. He believes that the disease is due to chemical changes.

Dr. C. G. Grulee, Chicago: It seems to me that there is another side to this proposition—the prime factor, the one thing necessary for such a theory to be realized—that this protein must be absorbed through a deranged gut. What causes the derangement? In answer to Dr. Johnson, I may say that Dr. Finkelstein's ideas have received a serious jolt in the last year. Bendix and Samelson have shown that the injection of sugar solutions made from freshly distilled water does not produce a rise in temperature when injected subcutaneously, while, if the distilled water used is old, a rise in temperature is produced. This, although not yet printed, has also been confirmed in Finkelstein's own clinic by a young Chicago physician, Dr. Grace Meigs, in regard to salts. This does not mean that we cannot produce a rise in temperature from the ingestion of salts or sugar by mouth. In fact, it has been shown definitely that those experiments are correct; that

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a certain amount of salt or sugar does cause a rise in temperature. The only way in which the seeming conflict of these two propositions can be explained is that the sodium salts or sugar, when given by mouth or rectum, causes a change which allows absorption of some product from the intestine and in that way produces a rise in temperature and symptoms. They have not determined the matter definitely, but have reason to believe that the product in the stale distilled water is not bacteria; but that does not make any difference, nor does it combat this theory of Dr. Vaughan's. It seems to me that, while there are many points that will have to be worked out, we may be on the right track in regard to the causation of summer diarrhea.

There is protein in the intestine other than casein, other than the protein ingested from food, which may be active in the mucus. This has not been accorded the place by pediatrists, which it perhaps deserves, as the source of putrefaction. This may cause a great deal of trouble. As we go further with Dr. Vaughan's idea, we may find that it is absorption of the mucus unchanged.

Dr. Louis Burckhardt, Indianapolis: Dr. Vaughan stated that some food is harmless at one temperature and harmful at another. In our city we have a first-class milk-supply. Our milk commission has controlled it. We have taken sample tests in the laboratories, and the last three tests came back with a count of three hundred. In spite of that, the board of health proposes to demand pasteurization of the milk. I am much interested to hear Dr. Vaughan's idea about the advisability of compulsory pasteurization of milk which has been produced under all the precautions.

Dr. H. W. Cheney, Chicago: Dr. Vaughan did not suggest it, but I wish to ask if this protein poison he speaks of is not the same as that present in horse-serum given therapeutically, which causes death in animals in which it is sometimes used. From a clinical point of view we can agree that some of the cases of summer diarrhea are caused by protein poison, but we know that a large proportion must be caused in other ways, because we can cure these patients feeding skimmed milk, a protein-rich food.

Dr. George S. Cattermole, Boulder, Colo.: In Colorado we have few cases of summer diarrhea. I believe the reason for this is our low mean summer temperature. Our nights are cool. Probably for this reason we are free from the intestinal disorders in infants that are so common in hot climates. So-called "cholera infantum" is almost never seen in Colorado.

Dr. T. C. McCleave, Berkeley, Cal.: I understood Dr. Vaughan to say that all protein poisoning is the same, the symptomatology depending on the localization in the body. In the exanthematous diseases, in which specific manifesta-

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tions appear so largely in one organ, the skin, how does he explain the occurrence of the different characteristic eruptions under his theory?

Dr. Victor C. Vaughan, Ann Arbor, Mich.: It is evident that the presence of other constituents besides proteins in the food influences to a great extent the absorption of proteins from the intestines. This absorption is largely a question of osmosis. It is altogether probable that one dilution of sugar favors the passage of the protein into the blood, while another dilution would have the opposite effect.

I am glad that this question concerning scarlet fever was brought out. As I have stated, experimental fever can be induced in animals by the subcutaneous, intravenous and intra-abdominal injection of any kind of protein. These proteins have their predilection places in the body, where they accumulate. This is true of both living and dead proteins. The pneumococcus protein accumulates in the lungs; the typhoid protein in the mesenteric lymph-nodes and in the spleen, and the proteins which are concerned in the exanthematous diseases accumulate in the skin and submucous tissues. We have shown positively by experiments in the laboratory that white of egg injected into the circulation of a rabbit soon disappears from the blood. Later it may be found in various tissues. White of egg injected into a rabbit intravenously accumulates largely in the skin and in the walls of the intestines. It is also found in the kidneys. Not only does the foreign protein disappear from the blood and become diffused through the tissues, but it also carries with it an appreciable amount of the normal proteins of the blood. The symptoms of one disease differ from those of another largely on account of the organ in which the foreign protein is deposited and in which it is disrupted. The most skilful physician cannot distinguish by the symptoms alone between a meningitis caused by the meningococcus and one caused by the pneumococcus or a third caused by the typhoid bacillus. In all acute infectious diseases the poison formed is one and the same thing. It is the protein poison that kills in all these diseases.

The question has been asked concerning the pasteurization of milk. I should prefer to feed a baby on pure rather than on bad milk, on milk with a low bacterial count than on that with high bacterial count, even if the bacteria are not pathogenic. Bacteria, whether they are pathogenic or non-pathogenic, split up the proteins of milk and probably render them absorbable without complete digestion. The exact relation of cholera infantum and the other forms of summer diarrheas to anaphylaxis has not been made out with certainty. I am, however, of the opinion that the absorption of partly digested proteins has much to do with the summer diarrheas of infancy.

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THE NATURE OF ILEOCOLITIS FROM AN ETIOLOGIC POINT OF VIEW

C. G. GRULEE, M.D.
CHICAGO

The term "ileocolitis," while originally a pathologic designation, has become distinctly clinical. No one, I think, will assert that all cases which show the clinical manifestations of ileocolitis are necessarily combined with the pathologic lesions which the term implies, and certainly not with the severe pathologic lesions which are commonly associated with it. It seems to me that the term ileocolitis conveys to the mind of most persons the clinical picture which follows in from twenty-four to forty-eight hours the acute stage of intoxication of alimentary origin. This period represents a symptom-complex which is more or less regular. The stools, while not so frequent as during the acute stage, contain much mucus and often blood. The temperature ranges usually between 99 and 101 F., prostration is great and the tolerance for food is markedly reduced.

It is this stage of acute attacks of summer diarrhea which offer so much difficulty in the solution of the problem and its cause. In general, three or four processes have been mentioned as possible causes of this condition.

The first is bacterial infection, which has received the greatest prominence at the hands of American pediatricians. "Bacterial infection" here does not mean the intoxication produced by decomposition products, the result of bacterial action on ingested food or its derivatives, but direct infection of the system through the

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intestinal wall. In this connection, two organisms have been more prominently mentioned, the *Bacillus dysentericus* of the Flexner type, and the streptococcus, and in by no means all of the cases are these two organisms found.

In order to explain the condition as a result of bacterial infection, it has been necessary to resort in some cases to the expedient of assuming that the *Bacillus coli* suddenly becomes pathogenic and causes the symptom-complex which is known as ileocolitis. No one, I think, will deny that no one organism is found in all these cases, nor that exactly the same clinical picture is produced regardless of the organism found in the gastro-intestinal tract. One should make the exception here of certain epidemics which have been reported from time to time, such as those caused by the Shiga bacillus, *B. pyocyaneus,* etc.

The chief reason for claiming pathogenicity for these organisms has been agglutination reactions in the blood. Whether agglutination can be regarded as direct evidence of the pathogenicity of that organism for a patient from whom the blood was obtained seems to me rather doubtful, since we know that such organisms as *B. coli* are agglutinated by normal blood in dilutions as high as 1 to 50. The mere presence of organisms of a doubtful pathogenicity in the feces and intestines of a child sick with ileocolitis certainly cannot be regarded as direct proof of the pathogenic action of these bacteria in a given case, and much more evidence must be brought to prove any such proposition.

The second process also has to do with bacteria, but in a decidedly different manner. In this process the bacteria act as agents decomposing the bowel content — more likely the proteid content of the bowel. The decomposition product thus formed would be regarded as toxic, and such product would produce this condition.

The theory of Kendall is certainly an ingenious one and deserving of our closest consideration. His clinical

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results, however, are not convincing and it is possible that his failure to obtain altogether satisfactory results may be explained by the fact that the bacteria producing the toxins continue their action below the point where the sugars are ordinarily absorbed and hence have the protein content of the bowel for the medium in which to grow. It should also be borne in mind when speaking of the protein content of the bowel that in the lower bowel this is composed in only a small part, if at all, of the proteid of the food, and there is no reason to think that the decomposition of the mucus produced in the bowel may not be carried on by the intestinal bacteria to as great a degree as is decomposition of the proteid. In this way it is easily conceivable that carbohydrates may increase the proteid content of the lower bowel.

Recent experiments have shown that volatile fatty acids, the product of fat and sugar decomposition, may be distinctly irritating to the intestinal mucosa, one result of which would be the production of mucus.

The third process is that of a metabolic disturbance produced by absorption, either of too great quantities of normal elements, or of elements which, as absorption products, may be regarded as abnormal. The chief advocate of this idea is Finkelstein, and his opinion, in part, is based on the fact that oral or subcutaneous administration of sodium salts or of sugar will produce a rise in temperature. Within the past year the experiments of Samelson, Bendix and Meigs have tended to disprove this theory. They were unable to produce a rise in temperature with sodium chlorid and sugar solutions made up from freshly distilled water. In other words, freshly distilled water did not contain some element which was present in the distilled water which had stood for some time, and this element, at present unknown, was responsible for the production of fever. It might be stated that this element in all probability does not consist of bacterial bodies.

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While it has not been possible to produce rises in temperature with the salts and sugars when administered subcutaneously, it has never been disproved that salts and sugars when taken by mouth will produce such rises in temperature, but we must remember that in this case the proposition is an entirely different one. Here the sugars and salts traverse the alimentary canal and are subject to the action of the intestinal ferments and bacteria. It may be that in this instance only more favorable mediums for the growth of intestinal bacteria and the production of toxins are produced. Whatever may be the cause of the fever in this instance, one cannot feel the same security in ascribing to the salts and sugars the metabolic action which many of us have regarded as the potent cause of summer diarrhea.

The fourth cause which may enter into this, but which probably affects rather the initial stage than the more chronic process, is heat. Just what position heat takes as an etiologic factor in summer diarrhea is by no means definitely determined. It seems reasonably certain that some cases of intoxication are really cases of heat-stroke, but further than this it is impossible to go at the present time.

It seems to me that in the present state of our knowledge it is impossible to decide definitely between these various processes, nor is it possible to assert whether or not the condition is due to the absorption of endotoxins of various bacteria unchanged through the intestinal wall. The fact that the clinical picture does not depend on the type of bacteria found in the intestinal content speaks strongly, to my mind, against the assumption of bacterial infection as a cause of the condition.

The disturbed metabolism as produced by the absorption of sugar unchanged through the intestinal wall is, in light of recent experiments, of rather doubtful value. A more convincing proof will have to be brought forward before it can be accepted as a cause of the condition.

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This leaves us with little more than a theory, a theory championed most ably by Czerny and Keller in the discussion of the nature of "toxicosis," a theory which has little to support it in the way of experiments, but against which, at the same time, few objections can be raised.

I am disregarding heat because it certainly cannot be regarded as a prime factor in the later stages of ileocolitis. In considering the condition as an intoxication produced either by absorption of endotoxins or of decomposition products of bacterial metabolism, one must take more account than has been done in the past of the intestinal mucus as a medium for bacterial growth and the source of decomposition products. Theoretically, at least, it would seem that any factor which tends to increase the flow of the mucus, whether in the small or the large bowel, should, if possible, be eliminated.

Perhaps of all the foodstuffs the protein is least irritating and the easiest of absorption, and for this reason, if for no other, it would seem that when food is to be given to infants suffering with ileocolitis, a properly dosed protein food should be the first selection.

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ABSTRACT OF DISCUSSION

Dr. R. M. Smith, Boston: I am not yet convinced that bacteria do not play a part in the etiology of ileocolitis. I saw a large part of the clinical work on which Kendall based his conclusions. I feel sure that there is truth in his contention that different types of bacteria contribute to the variety of forms of ileocolitis. The different response to different forms of treatment seem to me significant. The response is such as we would expect to receive if different bacteria played a part in the etiology. To illustrate, the cases of the group in which we are in the habit of assigning the dysentery bacillus as a cause respond favorably to the feeding of carbohydrates. We use sugar solution for this feeding. On the other hand, cases of ileocolitis in which the gas bacillus is the etiologic factor do badly when fed with carbohydrates, but do well when fed on a high protein diet. When the food furnished the individual is unfavorable for the development of the particular

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bacilli present, improvement in the patient is noticed. It seems to me that this different response to different treatment on the basis of a possible difference in bacterial etiology must be considered in this discussion.

Dr. E. W. Mitchell, Cincinnati: We should go away with a mistaken idea if we were to understand that bacteria did not play an important part in intestinal diseases. From the clinical side we have abundant evidence of the great importance of clean milk. By that I mean milk which has a low bacterial count. In my own city there has been an enormous change in the number of cases of sickness in babies in the summer time. I remember well when we attributed ileocolitis and other forms of intestinal trouble to the heat of the summer. Our summers have not changed in temperature; our hygienic conditions have changed for the better; people have learned much about the care of infants, but most important of all, there has been an enormous improvement in the cleanliness of our milk-supply. Those infants whom we feed throughout the summer on certified milk seldom show intestinal disturbance if they have ordinarily good care and if the milk is properly cared for at home. The remarks of Dr. Grulee are exceedingly suggestive as to the probable explanation of those cases which occasionally occur in children receiving a pure milk or children on the breast, and his explanation of why Finkelstein's theory would work out is suggestive. We all have had the experience that when we do use a fat-free milk we have far better results in the prevention of intestinal disturbances and in their more rapid cure. Perhaps Dr. Grulee also suggested a reason for this. In cases of intestinal intoxication it has been the fashion to give white of egg or albumin-water, with sometimes the result of symptoms suggestive of albumin poisoning.

Dr. J. L. Morse, Boston: I feel, as Dr. Smith has said, that our experience in Boston shows that different cases respond differently to treatment. What cures one patient will not cure another. On that basis we do feel that the bacterial etiology is important. It seems to me impossible to draw any absolute conclusions at present because our evidence is not as yet absolute. There is evidence on all sides. How to evaluate that evidence and what conclusions to draw from it has to be a good deal a matter of individual judgment and individual experience. I cannot quite agree with Dr. Grulee that the bacteria do not play an important part.

Dr. Clifford C. Grulee, Chicago: I did not say that bacteria do not play an important part in summer diarrhea. What I did say was that I do not believe that specific bacteria play an important part in summer diarrhea. The bacteria which are frequently accredited with being the cause of summer diarrhea, and especially the dysentery bacillus of the type ordinarily found — that is, the Flexner type, not the

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Shiga — has not been shown to be especially pathogenic. So far as I have been able to determine there have been a few experiments which have shown the pathogenicity of this bacillus to animals. The antidysenteric serum has proved a flat failure in the treatment of summer diarrhea of infants. The differences in opinion among Dr. Morse, Dr. Smith and myself are differences based on clinical results which cannot be eradicated unless we see the same set of cases. Other men have taken up the work of Dr. Kendall, and I think that I am fairly familiar with that work. I have heard him explain it himself. Others have had no results from the giving of sugar in cases of dysentery. One man followed this out clinicially and did not get the results that had been reported by Kendall. This is easily explained. One can never obtain as good results with a certain method as the originator of that method obtains. In the present state of our knowledge the only evidence of any value that we have for any specific bacterial cause for summer diarrhea comes from the work of Kendall, which is based on the clinical results obtained from different methods of feeding. As yet these results can scarcely be regarded as conclusive evidence.

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INJURIES PRODUCED BY STARCH

I. A. ABT, M.D.
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The *Mehlnährschaden* of the Germans, or the "starch injuries," have been extensively discussed in the German literature, especially in the writings of Czerny and Keller, and the Breslau school, and elaborated by the studies of Risel and Rietschel. To the genius of Czerny and Keller we are indebted for the conceptions of injuries to the organism produced by fat and starch and overfeeding with milk. These studies gave a great impetus to investigations of injurious effects produced by the component parts of milk. Thus, the ill-effects from excessive feeding of sugar, salts, starches, proteins and fats have been extensively studied.

In this brief clinical report I desire to review the work of the German authors on the starch injuries and to recite personal experiences. I feel it necessary to acknowledge that no original contribution is being offered, and feel that sufficient excuse for the publication of this report lies in the fact that few, if any, cases have been reported in the English literature.

This condition known to the Germans as *Mehlnährschaden*, seems to be little known in this country. Czerny and Kellar point out that starch in proper dosage is not injurious to the infant's organism. It has been suggested that nitrogen is better retained if small quantities of starch are added to the food. If it be given in excessive quantities for an extended period of time, particularly if it be used without the addition of milk, it may lead to disastrous results.

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The condition arises in young infants who are fed starch preparations without the addition of milk. More frequently it is seen in infants who have suffered from a gastro-intestinal disturbance and for whom the physician has ordered a starch water, either barley or rice or oatmeal, and when this has been continued without the addition of milk, usually without the knowledge of the physician, for a long period. The pathologic condition which results from overfeeding of starch is due probably to an actual food deprivation measured in calories. The starvation is particularly due to the insufficiency of protein, fat and salts in the diet. The salts contained in the starch food as well as the starch itself, tend at times to combine with water in the tissues, leading to water retention. This causes the infant to appear plump, though pasty and pale. In the meantime, the normal production of antibodies in the organism is diminished and the resistance of the infant against infection is lowered, and secondary infections are likely to occur. Dyspeptic conditions with diarrhea and loss in weight result. If the condition of inanition continues for some time, fermentation and inflammation and loss of function of the alimentary tract are inevitable. This latter condition will explain the atrophic type of starch injuries to be referred to again.

The disease presents itself in three main types. Very frequently the little patients appear plump and are thought to be unusually well developed.

1. In cases in which there is a deficiency of salt without retention of water, complicated by diarrhea, the atrophic type presents. The muscles are hypertonic, the tissues dry, the skin and mucous surfaces are pale and the abdomen is distended.

2. The hydremic type occurs most frequently in cases in which salt is added to the starch preparations, or in which a small quantity of milk is used in the food.

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These children remain stationary or increase in weight; sometimes the increase is enormous. If considerable milk is added to the diet at this point, the infants lose rapidly in weight. If the starch diet is resumed an increase in weight occurs. Gradually they present an edematous, pasty appearance, due in part to retention of water, and in part to accumulation of subcutaneous fat. The musculature is doughy on palpation; the abdominal wall and lower limbs become edematous, not due to nephritis but to a condition of simple hydremia. Owing to the diminution of the natural immunity the infant is predisposed to infections, such as bronchopneumonia, pharyngitis, otitis, abscesses and phlegmons. Czerny mentions that xerosis of the conjunctiva and cornea with sequelae are not infrequent and the condition usually indicates a fatal termination. He notes that ophthalmologists recognize the fact that infants with this eye disease seldom survive.

3. The hypertonic form is the most infrequent type. The muscles become rigidly contracted and are stiff and board-like. On account of this condition the spine is like a rigid tube, the occiput is bent backward and is bored deeply into the pillow, the arms and legs are adducted, the forearms are flexed at the elbow and the joints offer resistance to passive motion. In the severe types rigidity is so marked that the patient appears like one in catalepsy. Galvanic excitability with pronounced symptoms of tetany may be elicited. The bowel movements are usually yellowish-brown and formed, and frequently show starch with the iodin test. In the severer cases in which fermentation has occurred the stools may become thin with a penetrating sour odor.

PROGNOSIS AND TREATMENT

The dangers resulting from starch injury are most frequent in young infants. Starch can be digested by young babies, but only in small quantity. The mortality in these cases is high. The greatest danger exists

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on account of the loss of the natural immunity and lowered resistance, rendering infections more possible. The atrophic form seems most amenable to treatment and offers the best prognosis.

The treatment consists in the administration of milk containing a moderate quantity of fat. Carbohydrate food, such as buttermilk mixtures, malt soup and cereal decoctions should be avoided. Best of all is breast-milk. Even with the use of breast-milk one should consider the damaged tolerance of the organism and small quantities frequently repeated should be administered; first 2 ounces, 6 ounces, 10 ounces per day increasing gradually. Even with the use of breast-milk the cure will not be rapid. There may at first be loss of weight. The period of repair may be protracted, the damaged normal immunity may be slow to return and the use of the breast-milk should be persisted in for a considerable period, at least until the patient shows increase in weight. If breast-milk is not available, it has been suggested to use small quantities of undiluted cow's milk, 1 to 2 ounces per day, gradually increasing to 3 ounces and eventually to 6 ounces; finally by gradual increase to 10 or 12 ounces. The deficient quantity of water in the food may be counterbalanced by giving tea or water sweetened with saccharin. Or the milk may be used diluted with water, small quantities being frequently repeated. In some cases top-milk mixtures might be indicated, though the infant's tolerance for fat should not be exceeded.

CASE 1.—A. C., aged 10 months, admitted into my service in the children's ward of the Michael Reese Hospital, July 27, 1912. The baby was one of twins. She was breast-fed for the first month; then she was put on milk and Mellin's food until 4 months old, when she began to vomit her feedings. The mother was advised to try strained oatmeal gruels without milk. She instituted this plan of feeding until the present time. During the past week the patient has had diarrhea; first three or four yellow slimy stools and after the administration of calomel numerous green stools. She has

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not vomited, is very restless, cries a great deal and sleeps poorly. There is no history of previous illness.

Patient has six brothers and sisters, all living and well. When she was admitted into the hospital it was noticed that she appeared pasty, somewhat swollen. Her face was puffed and on palpation the surface of her body gave a doughy sensation. The eyes showed cloudiness of both corneae. On the left eye over the conjunctiva a large vesicle occurred. In the right eye several small vesicles were present. Marked photophobia, increased lacrimation and circumcorneal injection was noted. There was no purulent discharge. The bony thorax showed the presence of a well-marked rosary and a flaring of the costal arches. The abdomen was somewhat depressed. There appeared to be a loss of subcutaneous adipose tissue and the skin was harsh and dry. The abdomen was covered with numerous macular discolored areas varying in size from a pin point to a small bean, many tending to coalesce, showing a distinct bluish color—a diffuse purpura hemorrhagica. The photophobia tended to increase, and the cloudiness of the cornea, particularly of the right eye, was more distinct. There was marked keratitis in both eyes. During the first few days the infant's temperature was rather high, reaching 104 F. The leukocyte count was 33,500, with 82 per cent. neutrophils, six small mononuclears, eight large mononuclears, four eosinophils, and hemoglobin 62. On admission the child was greatly prostrated, rapid pulse and slightly accelerated respiration.

The infant at first was given a tea diet for twelve hours; then was placed on dram doses of breast-milk every hour and when an improvement in the appearance of the child was noted 6 drams of breast-milk were given every two hours. On the fifth day after admission the child was given 6 drams of skim-milk and 6 drams of breast-milk with 2 grains of citrate of soda, and 4 ounces of water every three hours. The baby lost 4 ounces in weight during the first several days of this treatment. On the fourteenth day after admission to the hospital the breast-milk and the skim-milk were increased to 2 ounces of each and 2 per cent. of *Nährzucker* was added to the diet. On the thirty-third day after admission she had shown considerable improvement and meat broths with vegetables were added to the diet. At the same time mashed carrots and baked potato were given without bad results.

An abscess appeared on the back after the child had been in the hospital for six weeks, which required incision and drainage. Later on a somewhat generalized furunculosis appeared, which seemed to improve with treatment by staphylococcus vaccine. One month after admission the patient showed an abundance of pus in the urine with elevation of temperature and considerable constitutional disturbance. The corneal opacity had entirely disappeared. The baby was play-

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ful, happy and active. She enjoyed her food, was gaining in weight, the purpuric spots on the abdomen slowly though constantly showed marked tendency to fade.

The child was discharged on Dec. 23, 1912, after fifty-nine days of treatment, showing a marked improvement.

CASE 2.—This infant, also one of twins, from the service of Dr. Julius Hess, weighed 7 pounds at birth. It was three months old on admission to the hospital. The first baby was breast-fed exclusively, while the second of the twins, which was the stronger at birth was breast-fed for the first two weeks, then given half milk and half oatmeal water for three weeks. Milk-sugar was added at the end of this time and the baby showed symptoms of indigestion and diarrhea. As it seemed quite impossible for the baby to take milk it was put back on the oatmeal water and this was continued for several weeks. After some time the bowel movements became thin and sour and frequent and she vomited very much. Emaciation was progressive and the baby weighed 6 pounds 2 ounces when she was three months old. The skin was dry and inelastic; the normal turgor was absent; the baby was atrophic and had a senile appearance. The breathing was rapid and shallow and the condition seemed desperate. The infant was placed on small doses of breast-milk and three weeks afterward was beginning to gain in weight and from this time on she made a rapid and complete recovery.

104 South Michigan Avenue.

ABSTRACT OF DISCUSSION

DR. F. P. GENGENBACH, Denver: I wish to cite a case that came to my attention about two years ago. The patient was a baby about 7 months old with acute gastro-intestinal disturbance. The doctor took the baby off its food and put it on barley-water. Unfortunately for the baby, the doctor became ill and forgot about it, and the mother left the baby on barley-water—nothing else—for two weeks. I found the baby with all the appearances of a child with marked nephritis. Its face was puffed up and the whole body edematous. The case might have been called one of acute *Mehlnährschadung*. Things were not so desperate as they looked, however. I took the baby off the barley-water, gave it a weak milk modification and it promptly recovered.

DR. F. W. SCHLUTZ, Minneapolis: A case of this kind has recently come under my care. The most pronounced symptom was the hypertonic contraction of the muscles of the extremities. Both hands were clenched and the nails buried deeply in the palms. This condition had existed for weeks. The child, for several months, had received only oatmeal or barley-water seasoned with salt. The extremities presented marked edema. Another feature in this case was the condition of the sensorium. The child gave the impression of idiocy. The sensorium was always clouded. The mother of

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the child had a 4-month-old baby which she was nursing. This fact made possible the therapy carried out in this case. The nursing baby was put on mixed feeding—part breast and part buttermilk. The sick child was given breast-milk, expressed from the mother, with supplementary feeding of buttermilk. The physical condition of the child has improved markedly. The mental condition and the hypertonicity remain unchanged. It will be interesting to see whether these conditions will improve or whether they are permanent changes.

Dr. Julius Hess, Chicago: I believe that there is an important lesson to be learned from these cases. Either we do not see many of them, or we lay the death to something else. I believe the latter is true. It is a common practice to tell the mother, if the baby has diarrhea, to put it on barley-water or rice-water, but I believe we are a little indifferent about warning as to the dangers of this, especially of its long continued and repeated use as an exclusive diet. Most mothers keep the baby on the cereal waters but a short time, but as soon as the baby has a loose bowel they repeat it. With each withdrawal of food and placing on cereal water exclusively, the convalescence is slower. I believe the lowered resistance, especially to general infection, is marked. It is our duty to warn against the complete withdrawal of food over any continued period.

Dr. L. Duncan Bulkley, New York: I do not know how many of you remember a paper I read many years ago before this section about a certain preparation for infant feeding which I called wheat jelly. I have used this for over twenty years in hundreds of cases. My opinion about all this trouble is that starch is generally not cooked enough. I use crushed wheat—the whole wheat. My directions are to take a teacupful of whole wheat, put it in cold water in the afternoon at 3 o'clock and boil for three hours. Then set it aside in a china vessel. Those of you who have eaten wheat which has been cooked over night know that it is sweet; a certain amount of diastatic fermentation has taken place. In the morning more water is stirred into it to make it quite soft, and it is boiled three hours more. It is then rubbed through a fine sieve with the bowl of a spoon. By scraping off what is under the sieve we have a jelly-like mass representing all the wheat, except the external silex coat.

Two or three teaspoonfuls of this wheat are put into the milk at each feeding. It passes readily through the nipple. In this manner we have extracted all the components from the wheat, including the nitrogen from the germ and the phosphates from the outer coating; we have then an entire wheat product, which nature intended we should take. For twenty years and more I have never used barley or any of the prepared foods, as I find that the wheat jelly is a perfect nutrient. I think that this is worth trying for the summer diarrheas.

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THE TREATMENT OF HEMORRHAGIC DISORDERS

THOMAS B. COOLEY, M.D.
DETROIT

Within the past few years, chiefly because of better knowledge of the physiology of the blood, much new light has been thrown on a group of diseases which have always aroused great interest, but whose pathology has been obscure, and whose treatment has been exceedingly unsatisfactory.

Of these diseases, the most important clinical feature of which is a tendency to uncontrollable hemorrhage, the most important to pediatrics are the so-called melena neonatorum, the purpuras and hemophilia; the last being, of course, equally evident in later life. Etiologically, we are still in the dark regarding all of them. Hemophilia shows two very important differences from the others. It is a permanent, not a temporary condition, and it is not characterized by spontaneous bleeding or oozing from the vessels, but merely by failure of the ordinary method by which a break in the vessel wall is sealed after it occurs. In purpura, in a number of the various conditions which are grouped under the vague term "melena," and in the hemorrhage of septicemia, which seem to be closely related, there is, on the other hand, a very pronounced tendency to spontaneous leakage from the vessels, as well as the failure of the blood-clot to form sufficiently to put a stop to this. In these diseases, moreover, if the outcome is not fatal, the hemorrhagic tendency usually entirely disappears, and the patients become wholly normal in this respect. These

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differences seem to me to be highly important in formulating anything like a satisfactory theory of the probable causes of the conditions and the rationale of treatment. It is wholly impossible, within the limits of a short paper, to analyze the voluminous literature of the past few years regarding the pathogenesis of hemorrhagic diseases. I shall try merely to present briefly the points important to my argument.

It has long been noted that in all these conditions the blood was often slow to clot, the clot when formed frequently was abnormally soft, and the failure of the clot properly to seal the vessel was generally believed to be the reason for the continuance of the hemorrhage. As the physiology of clot formation became better understood, a lack in the blood of some one of the essential elements was assumed to be the cause of this failure. Calcium, fibrinogen and prothrombin all being essential to clotting, a deficiency of any one of them might delay clot formation, while an excess of antithrombin would prevent thrombin formation even in the presence of sufficient prothrombin. The existence of thrombokinase is still in doubt, but there are those who believe that its absence will account for some cases. Definite proof of a deficiency of any of these elements was long lacking. Duke[1] in 1910 thought that he had shown a relation between a low platelet count and purpura and has emphasized this idea in further publications. The blood low in platelets is said to clot in normal time, but the clot does not contract sufficiently to seal the vessel. Schloss[2] found thrombin lacking in some cases of melena. Whipple[3] has found antithrombin present in septicemia, pro-

1. Duke, W. W.: The Relation of Blood-Platelets to Hemorrhagic Disease, THE JOURNAL A. M. A., Oct. 1, 1910, p. 1185; Jour. Exper. Med., 1911, xiv, 265; The Pathogenesis of Purpura Hemorrhagica with Especial Reference to the Part Played by Blood-Platelets, Arch. Int. Med., November, 1912, p. 445.

2. Schloss, Oscar M., and Commiskey, Leo J. J.: Spontaneous Hemorrhage in the New-Born, Am. Jour. Dis. Child., April, 1911, p. 276.

3. Whipple, G. H.: Hemorrhagic Diseases—Septicemia, Melena Neonatorum and Hepatic Cirrhosis, Arch. Int. Med., March, 1912, p. 365.

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thrombin absent melena, and fibrinogen deficient in hepatic cirrhosis. He suggests the possibility of classifying the hemorrhagic diseases according to which element is lacking. Calcium has not, I believe, been proved to be absent in any case of hemorrhage, though it is probable that the abnormal coagulability in jaundice may be due to relative lime deficiency. Sahli's[4] studies of hemophilic blood were interpreted as showing lack of thrombokinase, while Addis[5] thinks that he has shown defective activation of prothrombin.

It seems proper to believe, in view of this work, that temporary or permanent absence of some one of the clot elements is the usual cause of the persistence of hemorrhage in these conditions, and that different elements fail in different conditions, if not in different cases of the same condition. Whether or not we are also to believe with Whipple and others, that this is also the cause of the inception of hemorrhage, abandoning the older idea of some unexplained fault of the vessel walls, is another question. As Schloss has said, it seems impossible that a lack of any one of the clot elements should in itself account for a leakage of blood through a normal vessel wall; moreover, if this were the case, spontaneous hemorrhages should be common in hemophilia, where the blood abnormality is permanent. In spite of the lack of any definite proof, it seems to me that we must assume a diseased condition of the vessel wall as well as of the blood, except perhaps in those cases of the melena group where ulcers, thrombi or extreme congestion are present.

In accord with this idea of the failure of clotting as the cause of the persistence of the hemorrhage, treatment intended to supply the lacking element has very rapidly come into favor. The only inorganic blood-clotting element, lime, has been tried, but with little success. The other elements being all peculiar to the blood,

4. Sahli: Ztschr. f. klin. Med., 1905, lvi, 264; Deutsch. Arch. f. klin. Med., 1910, xcix, 518.
5. Addis: Brit. Med. Jour., 1910, ii, 412.

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blood preparations in one form or another are now being widely used in all of these diseases, and, if one were to judge merely by the reported cases, with almost invariable success. Normal animal serum was first used, chiefly that of the horse, sometimes that of other animals; the serums of the dog, bullock and goat being least in favor, because of toxic properties. Antitoxin has a number of times served in a pinch.

Lately, apparently owing chiefly to Welch's[6] very favorable reports, though he was not the first to suggest it, human serum has been used and highly praised by various American writers. Schloss injected whole human blood subcutaneously, believing it possibly advantageous to have all the blood constituents present. Very recently, precipitated, dried and powdered horse-serum, said to contain all the essentials to clot formation in at least normal concentration, and less subject to deterioration than in the liquid form, is being urged. Clowes[7] believes this to be more efficacious than fresh serum. Transfusion of whole blood has so far been reserved almost exclusively for use as a last resort in really exsanguinated patients, because, no doubt, of the difficulty attending ordinary methods of performing it.

Two things are very striking about the reports of successful treatment with blood or serum. One is the promptness with which the hemorrhage ceases, and the other, the relatively small amount used. Schloss, for instance, got prompt recovery after a single injection of 5 c.c. of whole blood, and most of the recoveries have been after injections of not more than 20 or 30 c.c. in all. Clowes reports the necessity of large amounts in one case of melena.

It is wholly impossible, at present, to gain any really accurate idea of the value of serum or blood injections or of the relative value of the different preparations. The tendency to report success rather than failure is well

6. Welch: Am. Jour. Med. Sc., 1910, cxxxix, 800; Am. Jour. Obst., 1912, lxv, 597.
7. Clowes: New York Med. Jour., Jan. 4, 1913.

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known, and many of the reports to be found are of single instances of success with any one of the preparations.

The reports of series of cases deal chiefly with melena. Swift and Green,[8] using rabbit-serum, had rather less success, apparently, than in the cases which were treated with gelatin and which they report in the same article. Schloss had much better results with human serum and whole blood, the whole blood being apparently more effective and in smaller quantities. Welch had remarkable results with human serum. Clowes' article deals with a series of very diverse conditions treated with solutions of serum powder. He seems to assume that there was lack of thrombin in all of these cases, though in some of them there seems to be no reason, except the persistence of hemorrhage, to suppose that a deficiency existed of any of the clot elements. In gastric ulcer and pulmonary tuberculosis, for instance, there is no need to assume failure of clotting in order to account for severe hemorrhage, though the cessation after serum injection might be held to prove that the coagulating mechanism was at fault.

I have found no reports of series of cases of hemophilia, but the isolated cases and small groups show almost invariable success with injections of any form of serum, or local applications of fresh or powdered serum to the bleeding spot. Prophylactic injections before operation are reported as successful by Clowes.

On the whole, the evidence from the literature seems distinctly in favor of human over animal serum, except for Clowes' series. This serum powder has only lately been available for extended trial, so that reports from other investigators are not yet at hand. My own experience with it in melena has been by no means so favorable as Clowes'. I have used it in two cases apparently with no effect whatever. In the first, which I have reported, the patient was saved by transfusion after serum powder and fresh human serum had apparently failed to check

8. Swift and Green: Boston Med. and Surg. Jour., 1911, clxiv, 454.

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the hemorrhage. Clowes' criticism (private communication), in this case that I did not use enough serum, may be justified. The second case was seen in the evening, shortly after the onset of hemorrhage, which was not at that time profuse. The baby received during the night 20 c.c. of serum powder solution and 20 c.c. of the father's serum, and died within an hour after the last injection of human serum, with symptoms of intrathoracic hemorrhage. Clowes seems not to believe that there is danger of anaphylaxis from the use of horse-serum. While I admit that this danger is slight, it seems to me sufficient to make human serum preferable, inasmuch as it seems to be at least fully as efficient.

There are at hand very few statistics containing the results of transfusion. Considering the very grave condition in almost every case in which it has been resorted to, it seems to me to have been more efficacious than any of the other methods. Several patients have been cured by it after serum injections had failed to check the hemorrhage.

Curiously little has been said by any of the writers concerning the probable action of serum or blood in checking hemorrhage. It seems to be taken as a matter of course that the blood preparation acts by supplying the deficient clot element, whatever it may be. The correctness of this assumption seems to me very doubtful. None of the clot elements are supposed to have a ferment action except the purely hypothetical thrombokinase. Definite quantities of each of the others go into the reaction, and it is difficult to suppose that the injection of relatively small amounts of serum or blood can possibly make up for any serious deficiency of any of them. It seems to me impossible that Duke can be right in supposing that the enormous increase in the platelet count reported by him after transfusion was the result simply of the addition of the platelets of the transfused blood. The amount of blood transferred by ordinary transfusion, which cannot be definitely measured, is certainly

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too small for this to be true. Admitting the existence and importance of thrombokinase, and assuming it to be present in any quantity in the injected serum, it might explain the cessation of hemorrhage in cases where thrombin formation is insufficient.

Another thing not satisfactorily explained by the idea of injecting a lacking clot element is that the patient is so often definitely cured. Not only is the existing hemorrhage stopped, but the tendency to bleeding disappears, and the blood soon shows normal coagulability. It is, I think, more reasonable to suppose that this effect is due to some stimulant action of the normal serum or blood on the blood-forming tissues or in the vessels. Esch has suggested that the bone-marrow is stimulated to the formation of thrombokinase. As we have no reports of the successful use of serum in cases in which any other element than thrombin was definitely proved to be lacking, this explanation might be correct. In the absence of more exact information, I prefer to suggest that the blood-forming tissues react to the stimulus of normal serum (or blood), by the formation of normal blood, and that possibly there is a reaction between normal blood and the vessel-walls, lacking in certain abnormal blood-states, which explains the complete cessation of hemorrhage. This hypothesis is vague, but something of the kind seems necessary to fit all the facts.

Such an assumption would give a reason for the apparent relative values of treatment by animal serum, human serum, whole human blood by subcutaneous injection, and whole blood by transfusion, which would not at all apply if the end to be obtained was merely to supply a lacking clot-element. Human tissues would naturally react better to human than to animal serum, to whole blood than to serum, and probably to whole blood put directly into a vessel than to the same blood injected subcutaneously, where it must partially disintegrate. I make this suggestion for what it is worth, admitting it

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to be pure theory, and incapable of demonstration with our present knowledge.

All things considered, whatever theory one may adopt, it would certainly seem, so long as we cannot determine at the bedside just what blood element may be needed, that there should be a wider use of direct transfusion in serious hemorrhage, because of its supplying for immediate availability all of the factors likely to be concerned; and that the transfusion should be early, rather than as a last resort after everything else has failed. The technical skill required has heretofore stood in the way of this, but recently there has been more than one suggestion of simple methods for accomplishing the operation. Crile's method is an improvement over direct suture or the older cannula methods, but is not readily applicable to the small veins of babies. Vaughan and myself[9] have described a method used by us which required no special apparatus, and which we have found to be really as easy as any intravenous injection. It consists simply in drawing blood from the donor's vein into a glass syringe and injecting it rapidly into the vein of the recipient. A small amount of physiologic salt-solution is drawn into the syringe before and after the blood, to prevent clots forming in the needle or the vein. The only requisite for success, apparently, is to have everything so prepared that one may work fast and keep within the time-limit of coagulation. We have ourselves practiced the method once since our report, in a case of septicemia, and Rowland (private report), used it without difficulty and with remarkable results in a case of severe melena with bleeding from the cord, mouth, and numerous areas in the subcutaneous tissues. Pope has recently described a method used by him on laboratory animals which he says is very easy. Freund, working with the anemias, has recently developed an improvement (unpublished), on our syringe method, by which he was able easily to trans-

9. Cooley, Thomas B., and Vaughan, Walter: A Simple Method of Blood-Transfusion, THE JOURNAL A. M. A., Feb. 8, 1913, p. 435.

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fuse 140 c.c. in a few minutes. The apparatus consists of a syringe connected by a Y and short rubber tubes, with two needles. At the branching of the Y is a two-way valve, and connected with the proximal branch of the Y is a tube, with stopcock, for salt solution. The needles are inserted into the veins of the donor and recipient and connected with the syringe. As the blood is drawn from the donor's veins about 10 per cent. of salt solution is allowed to mix with it. Dilution in this way with about 10 per cent. of saline solution prolongs the coagulative time considerably. The valve is turned and the diluted blood is injected into the patient. This procedure may be repeated, 20 c.c. at a time, almost indefinitely, with no apparent danger. Either of these syringe methods is as simple, and probably as safe as ordinary intravenous injection, and they have the additional advantage over ordinary transfusion methods of being quicker, as the positive suction and pressure of the syringe make it possible to transfer the blood much more rapidly. Moreover, it is possible, working by either of these methods, to interrupt the procedure at any time, and renew it at pleasure. With former methods, the difficulty of the operation and the sacrifice of the vessels which is involved, required one, in order to be sure of going far enough, to carry it often to very tedious lengths. The amount really necessary to be injected sometimes proves to be very small. Rowland, for instance, could see almost immediate results from 8 c.c., and was not obliged to use more, but with any other method he would have felt compelled to go a good deal further before breaking the connection between the vessels.

It seems probable, from a perusal of current medical literature, that human serum or blood is to be used, or at least extensively experimented with, in the treatment of a variety of conditions aside from those I have spoken of. Already they are being tried in the anemias, and in pediatrics there are favorable reports of human serum in grave nutritional disturbances. In all of these, it is

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possible that human blood injected intravenously might be of more use than serum. The purpose of this paper is to urge that simplified transfusion be given a more extended trial, especially in melena, which is one of the most serious conditions with which we have to deal; and that it be used as a first rather than a last resort.

CONCLUSIONS

1. Blood therapy of some kind is the best remedy we have for hemorrhagic conditions.

2. In hemophilia blood-serum seems to have a specific action so far as checking the hemorrhage is concerned. It may be used as a prophylactic measure, as well as to stop existing hemorrhage. Fresh human serum is probably to be preferred.

3. In purpura, melena, and other toxic conditions, in which various blood elements have been shown to be lacking, none of the serums are always effective, and there are good theoretical and clinical reasons for believing that whole blood should be preferred, not only to stop hemorrhage, but for a possible curative effect on the underlying disease condition.

4. Transfusion is not really a difficult procedure. It is deserving of extended trial—not as a last resort, but as the first treatment in any of the hemorrhagic diseases of toxic nature.

NOTE.—Since this paper was read, Freund has published a description of this method (*Jour. Mich. State Med. Soc.*, September, 1913, xii, 459).

Fine Arts Building.

ABSTRACT OF DISCUSSION

DR. V. D. LESPINASSE, Chicago: I have treated ten cases of hemorrhagic disease of the new-born by direct transfusion of blood. Some of these cases had been treated previously with various serums. Among the ten cases there were but two deaths; both patients were weak syphilitic babies. The cases were of all grades of severity from a comparatively mild to a very severe form. The most desperate case was that of a child born on Thursday at midnight. It started to bleed from the bowels on Saturday at 6 p. m.

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While being taken to the hospital the child vomited blood. It was transfused at 9 p. m., three hours after the discovery of the hemorrhage. While I was dissecting out the vein, the pulse stopped and the heart was neither audible nor palpable. Fifteen seconds after the blood began to flow into the internal jugular vein, the heart began to beat and the child recovered. The child was in the hospital one week and gained an ounce a day in weight. It is now alive and well. In transfusing these cases, I do not know how much blood is put into the vascular system. I transfuse until the children are red as in scarlet fever. When the operation is begun the babies are pale. If we allow the blood to flow in too rapidly the baby becomes bluish. If this occurs, the flow of blood should be stopped temporarily. When the baby's skin is a decided red, the anastomosis between the two vessels is cut and the vessels ligated. The effect on the babies is immediate and prompt. The hemorrhage stops at once and does not recur. I should like to emphasize the point that Dr. Cooley made, that transfusion is the best treatment for hemorrhagic disease of the new-born, and should be used early. I should also like to emphasize the fact that it is never too late to save a baby by transfusion, so long as there is a flutter in the heart; even if the heart has stopped for a few minutes, there is a chance to save the baby by a transfusion properly done.

Dr. H. M. McClanahan, Omaha: The remarks that I have to make are in the interest of the doctor in the small town who does not have modern facilities. I am glad that Dr. Cooley made the distinction between hemorrhage in the new-born and hemophilia. By hemorrhage in the new-born I believe he means spontaneous, not traumatic, hemorrhage.

Within the past year I have had five cases of hemorrhage in the new-born. In these cases I have recommended the use of antidiphtheritic serum, using a serum with a low number of antibodies. I have personal knowledge of three cases in which the hemorrhage was evidently sufficient to cause death, although I do not believe that these infants die from actual loss of blood, but from that condition which causes the loss of blood, and I believe that antidiphtheritic serum is an excellent remedy. It is not so good as transfusion, but in the small towns it is a good substitute.

Dr. Julius Hess, Chicago: The thing which has interested me most is the evolution of the medication in the hemorrhagic conditions. At first most of us used diphtheria serum without further thought. Following that a normal horse-serum was put on the market and we used that. Then came anaphylaxis. Then we used rabbit's blood, and eventually adopted the simple method of drawing a small quantity of a 10 per cent. sodium citrate solution into the barrel of the syringe, taking from 10 to 15 c.c. of blood from the

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vein of the mother and injecting the mixture directly into the vein of the child. Theoretically there are some reasons why we should not use sodium citrate. The results with this are as good as with horse-serum, and its use has obviated the dangers of anaphylaxis.

Dr. Ogden M. Edwards, Jr., Pittsburgh, Pa.: This is a serious condition which occurs in children at the beginning of their lives. The treatment at present is not satisfactory, as Dr. Cooley says. The use of serum and of some of the preparations of blood is the best treatment we have to-day. We shall never get the proper treatment for all of these cases until the etiology of these conditions has been discovered. We probably have a right to believe that these are different conditions, having one thing in common, spontaneous hemorrhages, and that there are different causes for the hemorrhages. If we could find these different causes we should get at the treatment better. Some hemorrhages may be due to infection, as has been suggested, but whether or not infection is the etiologic factor we are unable to say. Some of them are associated with syphilis, but we are not able to say whether or not that is the cause. Certainly we know that some of these children are not syphilitic.

The suggestion by Evarts Graham is interesting; that lack of proper oxidation existing in the mother before the child is born may play a part in the production of these conditions. He has brought about a change in animals experimentally by producing a lack of oxidation in the mother.

The etiology of these conditions is still unknown, however, and the treatment, therefore, will never be on satisfactory grounds until these hemorrhages have been differentiated according to their etiology. Some of the patients recover with serum and blood preparations, and some do not.

Dr. H. Lowenburg, Philadelphia: I should like to report a case of hemophilia in a 2-year-old child. His mother's brother died of uncontrollable hemorrhage following a slight injury, and his own brother succumbed to this same condition. This boy bit his tongue and had been bleeding for two days when I saw him. He was very anemic and restless. He responded promptly to two injections of horse-serum given at 7 p. m. and the following day at about 10 a. m. A point of interest that this particular case illustrates is that probably a permanent cure is not produced, because about five or six months later this boy suffered a slight trauma of the hip, which was followed by extensive ecchymosis. There was no break of the skin, and the ecchymosis was five or six weeks in disappearing.

Dr. C. Brooks, Pittsburgh, Pa.: I have for some time been interested in the technic of transfusion. Almost two years ago I suggested the use of a syringe to transfuse blood

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in infants. Since I located in Pittsburgh Dr. Edwards has spoken to me about some cases in which transfusion by the union of blood-vessels failed. I suggested that we try to work out some simple methods of transfusion. I am gratified to learn from the discussion here to-day that a good many simple methods have already been used. For instance, Dr. Cooley's method, in which he used a syringe with an ordinary needle and a method of drawing the blood in at one side, and throwing it out from the other with a "Y" shaped connection, is doubtless a good one.

Dr. Edwards and I have a method which we think more certain of success and more easy of use than any method yet discovered.

I think that all physiologists will agree that it is best to use for transfusion the normal blood. That is the least dangerous. Defibrinated or salted blood are departures from the normal. From theoretic considerations, however, the least objectionable salt to use for that purpose would be sodium oxalate. We have performed some experiments on animals, using this salt. It works very well. We draw into the syringe about 0.5 c.c. of a 1.0 per cent. solution of sodium oxalate and then draw the syringe full of blood. When this is done it will not clot. The needles which we have tried on animals are fashioned so that when once inserted they will not come out, neither will they cause laceration of the wall of the vessel.

I think that Dr. Cooley is right—we should not necessarily call in a surgeon to do transfusions. Those living in the country cannot always get a surgeon. They must go ahead and do the simplest transfusion possible. If they employ this syringe method, they will know how much blood they are transfusing. Some surgeons tell me that when they use the more complex methods they are not certain how much they transfuse, or indeed, whether or not they transfuse any blood at all. Apparently the right tendency is toward a simple method and toward the use of a syringe with the trocar cannula needle.

Dr. T. B. Cooley, Detroit: In regard to the blood necessary for transfusion, Dr. Lespinasse has said that in direct anastomosis methods it is advisable to transfuse babies until they are red. In the case reported, the child was practically dead when we started. We used altogether 20 c.c. of blood, with salt solution between the blood injections. We did not carry transfusion anywhere near the point of redness of the child. The child was still pale, but we could see a definite improvement; the pulse was becoming stronger and we felt safe in waiting; we knew that we could transfuse more blood at any time. The child did well. As regards transfusing blood slowly, we used injections of salt solution between the

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injections of blood. Maybe, if we had put in one blood injection after another, we should have gone too fast.

The point that I was especially anxious to make was that the doctor in the small town can do transfusion with one of these simple methods. It is a thing anybody can do who can dissect out a blood-vessel.

Nobody answered Dr. Hess' question about anaphylaxis. I have been unable to find any reports about anaphylaxis from the use of antitoxin in this way. Those of us who use diphtheria antitoxin extensively know that the proportion of anaphylaxis is slight. When horse-serum has been used in a large number of hemorrhagic cases, we shall probably hear of some cases of anaphylaxis.

There is one thing quite evident in regard to the etiology of nearly all of these conditions—they are the result of toxemia of some kind. Toxemia resulting from lack of oxygen is certainly a possible cause.

I was careful to except hemophilia from the conditions in which one gets a permanent cure.

I do not think that the addition of oxalate to the blood is necessary or desirable to prevent the blood from coagulating. If one can delay coagulation of the blood a little time, that is all that is necessary. If we are sure of our technic we can do this without any other assistance.

Dr. Brooks' needle is much like one which Dr. Vaughan and I made, although we decided that it was not absolutely necessary to have a special needle.

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TREATMENT OF INGUINAL HERNIA IN CHILDREN

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The elaborate classifications of inguinal hernia made by the older writers are no longer recognized. Direct inguinal hernias are now considered as abdominal hernias, and the division of the indirect into infantile, congenital and acquired has been given up because of the increasing evidence that all depend on the presence of a peritoneal sac which is present from birth.

Camper (1762) and Wrisberg (1779) concluded from dissections that inguinal hernias are due to persistence of a patent processus vaginalis (Hessert[1]). The anatomic studies of Zuckerkandl[2] indicate the same thing. Bittner,[3] has never seen any but congenital hernias. Universal acceptance of this view is still in the future, yet careful dissections and study of tissue removed at operation are sufficient to convince one of its correctness.

The common occurrence and especially the form of the sac have led Murray[4] and Hessert to this conclusion. According to the latter, the sac tends to obliterate at three points: the internal ring, the external ring and just above the testicle. In hernias there is usually a constriction at the external ring, indicating the tendency to close at this point. Even in the scrotal type, which extends nearly to the testicle, there is often a constriction in the region of the external ring. From

1. Hessert: Tr. Western Surg. Assn., 1910, xix, 173.
2. Zuckerkandl: Arch. f. klin. Chir., 1877, xx, 215.
3. Bittner: Arch. f. klin. Chir., 1894, xlix, 803.
4. Murray: Lancet, London, 1906, i, 363.

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the closed end of the sac a fibrous cord representing the obliterated sac can be traced toward the testicle. In hernias that have existed for a long time and have become large the constriction may be pushed downward from its usual position at the external ring. Dissection of the fibrous cord in such cases shows that there has been a displacement of the point of closure by a distention and sliding downward of the entire sac.

If gross dissection leaves one in doubt as to the existence of the fibrous remains of the peritoneal sac the point may be cleared up by microscopic examination of the tissue removed at operation, provided care is taken to preserve its spatial relations. This procedure furnishes the strongest argument in favor of a patent sac in all cases. Castenholz[5] adds the further convincing evidence that the sac is intimately united to the tunica vaginalis testis and to the cord. This union is so intimate as to preclude the possibility of its being formed after the origin of the hernia.

If the connective tissue at the point of union of sac and cord be examined microscopically, it is found to be made up of an interlacement of fibrils running parallel to the walls of the sac and continuing over the cord, but separated entirely from the other surrounding tissue. I have noted repeatedly that in less than five days after the appearance of hernias of sudden onset the union of sac to cord has been made up of fully developed fibrous tissue free from cellular infiltration. Obviously the union could not have taken place after the hernia developed. I saw the same condition in a person who had suffered from hernia in childhood, but had been free from it for eighteen years. Operation was performed only four days after the appearance of the hernia, but a sac was present, attached to a cord and clearly preformed. I have noted the same condition in hernias said to have occurred after traumatism. The comparison of the sacs of inguinal hernias to the sacs of abdom-

5. Castenholz: Deutsch. Ztschr. f. Chir., 1909, xcix, 257.

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inal hernias which were known to be acquired is convincing. These acquired sacs do not adhere so readily as those of inguinal hernia, and when adhesions do occur the connecting bands are not parallel with the sac wall as in inguinal hernia.

There is no exact information as to what proportion of infants retain a patulous processus vaginalis. In the new-born up to the tenth or twentieth day nearly all are open, while at the second year about half are closed on both sides. What proportion of these closes subsequently is difficult to say. Palpation of the internal ring in the course of laparotomies is not satisfactory, since the palpating finger is too large to enter a small opening. Yet even by this crude method a patulous canal can be demonstrated in perhaps 10 per cent. of cases. This percentage is great enough to account for the 6 per cent. of hernias which are supposed to afflict adult males.

It is certain that many infants who have never had hernias have patulous canals (Murray[6]). The explanation of this is not precisely clear, although several factors are plainly concerned. An abnormally long mesentery is necessary; a normal infantile mesentery could not permit the descent of the intestine in the canal. Some relaxation of the mesentery is necessary. Increased intra-abdominal tension such as results from constipation, straining at urination because of a tight prepuce, and constant coughing because of some pulmonary disease have been assigned as causes. Not infrequently a hernia is first observed in an illness during which the child cries a great deal.

The age at which hernias appear varies greatly. Strangulations in the earliest weeks of life have been reported, but usually they appear between the sixth month and the fourth year. They are rare between the sixth year and puberty. The infrequency of hernia during adolescence is explained by the protection which

6. Murray: Lancet, London, 1906, 1, 363.

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the canal receives from the developing muscles. The greater incidence of hernia after adolescence is explained by Castenholz on the ground that after puberty the person enters the struggle for existence and his more strenuous life brings about the hernia. That labor is more strenuous than the play of boyhood is not in accord with the experience of most persons. There is more probably some change in the inguinal canal due to the enlarged cord which facilitates the descent of abdominal contents.

DIAGNOSIS

The diagnosis of hernia in children offers no difficulty. The factors which cause confusion in adults are not present in children. Enlarged glands from rectal infection rarely cause a moment's hesitation. Venereal infection in children, so far as I know, does not cause confusion. The only question that arises concerns the contents of the sac. An undescended testicle often appears in the inguinal canal and may be unsuspected if the scrotum is not examined. Ovaries which may appear in the canal are usually diagnosed only at operation. Sometimes, however, their outline may be made out by palpation.

The subsequent course of a hernia is of great importance. In many instances the hernia having once appeared ceases to come down after the child becomes more active. Whether or not a sac that has once contained a loop of intestine or omentum may spontaneously close cannot be definitely stated. Why peritoneal surfaces become spontaneously obliterated during fetal life and soon after birth is unknown. It is not possible to prove that obliteration can be caused by any artificial means. It is maintained by many, however, that some do close spontaneously. The proportion cannot be even approximately stated, for it is quite impossible to keep these patients under observation. From the evidence at hand it is more likely that even when the hernial contents cease to descend the canal remains patent and

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is always ready to be filled whenever there is a weakening of the muscle or an unusual increase in intra-abdominal pressure. Stiles has demonstrated that in the so-called cures the sac remains patent and only awaits favorable conditions for the reappearance of the hernia. Kovacs[7] believes that it is but rarely if at all that a hernia becomes spontaneously cured.

If a hernia is allowed to go untreated, strangulation with its attendant dangers is always possible. Usually even strangulated hernias can be reduced after a time and death from strangulation is very rare.

PROGNOSIS UNDER TREATMENT

Treatment by means of the truss does not materially alter the course. The purpose of a truss is to retain the hernial contents within the abdomen and permit the surrounding tissues to regain their elasticity. There is nothing in the physiology or adaptive pathology of the peritoneum to warrant us in the hope that keeping the canal empty by means of a truss will cause the sac to become obliterated. That the pressure of the truss may cause sufficient irritation of the opposed surfaces of the sac to cause them to become adherent is of course beyond the possible. At the same time it is necessary to give respectful attention to the palliation of hernia by means of the truss. Two recent books on surgery in childhood favor this treatment within rather wide limitations.

It has been estimated that approximately 25 per cent. of patients treated with the truss are temporarily cured by it. That is to say, after being worn for several years the truss may be left off without a return of the hernia for a period of years. Subsequent history is difficult to obtain, for it is quite impossible to maintain a record throughout the patient's lifetime, and those who advocate truss treatment make no attempt to answer the question as to the future of the so-called cured cases.

7. Kovacs: Arch. f. klin. Chir., 1910, xci, 177.

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One meets, however, patients with hernias in later life who give a history of hernia in childhood, which had been apparently cured by a truss. One is therefore inclined to look on the curative value of the truss with skepticism.

The truss has been used for infants only a comparatively short time. It was formerly thought inadvisable because of the disposition to favor troublesome eczemas and even fatal gangrene. Ravoth,[8] as late as 1868, found it necessary to combat this view. As a matter of fact, however, if one entertains seriously the notion that a sac can be obliterated by the pressure of a truss, troublesome excoriations and even graver lesions are very likely to appear. The younger the child the more difficult it is to keep a truss applied.

Most practitioners content themselves by the use of the well-known skein truss. These more often hide rather than retain the hernia. In fat babies spring trusses are kept on with great difficulty and elastic trusses with perineal straps are apt to cause troublesome excoriations. In thin, emaciated babies better results may be obtained. After from three to six years, depending on the activity and build of the child, a truss can be made to retain the hernia with a certain degree of efficiency. The abuse of the truss comes in allowing the child to bear the discomforts of a steel band carrying a pad which rests somewhere in the region of McBurney's point. This gives a false sense of security and unnecessarily punishes the child, especially when a strong spring is used with the idea of obliterating the sac by pressure. The whole development of the child, mental and physical, may be retarded by a troublesome hernia or an ill-fitting truss.

Operative treatment has gradually come more into use in recent years. This is due to the more general appreciation of the anatomic relations of hernia and to a perfected technic in its radical cure.

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8. Ravoth: Berl. klin. Wchnschr., 1868, v, 470.

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Palliation is still largely practiced chiefly by two classes of persons; those who are opposed to operation by principle and those who employ palliation from necessity, either because the parents do not consent or because facilities are not available. Those who oppose radical cure from principle may be divided into two classes, those who still believe that hernia may be cured by the truss and those who have met disaster in radical treatment. Among the latter may be mentioned Grunert,[9] who had three deaths in thirteen patients. He advises against operation during the first three years.

Palliation will still retain a large number of advocates because of necessity, especially in regions in which operative facilities are not available.

A considerable number of operators advise operation in all or nearly all cases. Perhaps Stiles[10] has spoken with the clearest vision and the greatest authority. He operated on 360 patients. Of these, one died of infection, one from chloroform, one from gangrene from too vigorous manipulation made elsewhere and two from causes not given—five in all. There were four recurrences. Kovacs[7] had 232 cases with one death from imperfect technic. In 144 cases traced there was one recurrence. Castenholz[5] advises operation in all cases, because it is the only means of cure and because by curing the trouble in childhood when time is worth but little, the patient is spared incapacitation in later years when the struggle for existence is on. Pfaehler[11] advises operation in all cases. He had one recurrence and no deaths in forty-six cases. He treats them as ambulants, the patient being brought to the hospital only for operation and returned home immediately afterward.

More conservative are Coley and Bull,[12] who, after an experience of 1,500 cases, advise operation after

9. Grunert: Deutsch. Ztschr. f. Chir., 1903, lxviii, 518.
10. Stiles: Brit. Med. Jour., 1904, ii, 812.
11. Pfaehler: Deutsch. Ztschr. f. Chir., 1912, cxvi, 543.
12. Coley and Bull: Med. Rec., 1905, lxvii, 401. Coley, William B.: The Management of Hernia in Infancy, with Results of Operative Treatment, THE JOURNAL A. M. A., Jan. 14, 1905, p. 112.

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truss treatment of one or two years. Earlier operation is done if there are frequent strangulations, if the hernia is irreducible or complicated with hydrocele. In femoral hernias operation is always done. Buford[13] likewise does not operate before the fourth year, except in emergencies. Maass[14] is likewise disposed to be conservative because of the dangers of the operation. He operates only when the hernia increases in size, when there is troublesome eczema or when the parents are unable to care properly for the child. The reason for this conservatism is the danger of chloroform and the danger of injuring the vas and the vessels of the cord with subsequent necrosis of the testicle. Coley agrees with Maass that the operation is more difficult than in adults.

The point of view of those who operate is, therefore, largely influenced by results achieved. Those who advocate operation must answer both for deaths and for failures, and these factors are both still large enough to dampen enthusiasm. Hernia in children rarely causes death and it devolves on the advocates of operation to show a mortality less than that of the untreated cases. Statistics are not sufficient to determine the point. Stiles[10] estimates that 7 per cent. of all children visiting the clinic (Polyclinic at Edinburgh) have hernias. His mortality was nearly 1.5 per cent. Castenholz[5] estimates the mortality at 0.3 per cent. Those who can show a mortality so low as this are justified in advocating operation, without reserve.

That operation *per se* is without danger is evident, and it is only the surgical accidents that make us pause. Thrombosis after operation has not been observed. Death from chloroform, so often complained of by the Germans, can be avoided by giving ether. Lung complications and intestinal disorders may appear merely coincident with, or because of, the operation; it is impossible often to say which. Injury to the vas and

13. Buford: Illinois Med. Jour., 1910, xviii, 17.
14. Maass: Deutsch. med. Wchnschr., 1901, xxvii, 148.

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cord is purely a technical error and should not occur, at least not more than once. The surgeon's obligations resolve themselves into a question of asepsis. The delicate skin does not admit of rigorous cleansing and the difficulty of sterilizing a urine-soaked skin is considerable. The greatest difficulty, however, is in keeping the wound clean after operation. Collodion dressing is often inefficient to protect the region. It is this difficulty in keeping the parts clean that has caused many operators to prefer to wait until the child has reached an age when it can cooperate in the matter of cleanliness. After the child has attained the age of 3 or 4 years, spontaneous cure or disappearance for any considerable time is unlikely After this time the child is more likely to be conscious of his handicap, and the desirability of a permanent cure is proportionate to the degree of discomfort and the sensibility of the child. The operation is entirely safe and the cure nearly certain.

The type of operation likewise is subject to differences of opinion. If the patent canal is the only factor, those who advise a simple ligation of the sac are right. Castenholz closes the canal only when it is wide. Stiles believes that the Bassini technic is not needed, because of the shortness of the canal. Coley's experience is in favor of the regular Bassini.

Ambulant treatment is readily applicable and places the operation within reach of many who could not otherwise avail themselves of it. Castenholz sees his patients only after a week or ten days when it is desired to remove the stitches. This shows a confidence in the integrity of the dressings which is not warranted by the experience of most operators; but if the dressings can be observed from time to time Castenholz' method is a very efficient one. My own experience has been that the ambulant treatment is more satisfactory in children after four years than in infants. In the latter, no matter how careful the dressing, trained supervision is required.

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SUMMARY

1. All inguinal hernias in infants are due to persistence of the processus vaginalis.

2. Inguinal hernias are not cured by the truss and rarely recover spontaneously.

3. Surgery is permissible in all cases if the facilities are of the best and the operator is skilful. Operation is urgent if the hernia protrudes persistently or if the child is annoyed greatly by retentive appliances which lessen his activities or produce excoriations. Operation is demanded if the hernia is irreducible or strangulated.

4. Because of the difficulty of maintaining an aseptic field after operation it is wise conservatism to wait until the child has reached such an age as will enable him to lend his cooperation, that is, until about the fourth year.

5. Palliation is demanded when organic disease is present or adequate facilities for operation are not at hand.

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ABSTRACT OF DISCUSSION

Dr. Coleman G. Buford, Chicago: The treatment of hernia of infancy is nearly always considered and discussed with that of children in general and consequently there is great confusion. Hernia in infants is one thing, while that in older children is another. I shall try to limit my remarks to the former.

I admit that a large number of cases are congenital; but I have been surprised at the large percentage of supposedly congenital cases showing anatomic evidences of being acquired hernia.

The question of whether or not to operate on uncomplicated inguinal hernia in infants was definitely settled in America by the writings of Bull and Coley, based on almost unparalleled experience and statistics. Their conclusion was that children under 4 years of age should not be operated on for uncomplicated inguinal hernia until truss treatment had been used intelligently for four years. We have endeavored to follow this rule in my service at the Children's Memorial Hospital; but in older children we are inclined to shorten the period of truss treatment.

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Babies should not be operated on as a routine because of their susceptibility to infectious diseases when brought among other sick children; diphtheria, scarlet fever, measles and erysipelas appear too often when least expected. Again, male babies are especially liable to wound infection when herniotomized, because of the wound's close proximity to the genitals and anus. Babies are susceptible to the damages of pyogenic infection, even stitch-hole abscesses may easily be the indirect cause of the death of an infant.

I usually refuse to operate on children until (1) they are of such an age and state of intelligence as to aid in keeping their dressings and bedding clean; (2) a complication not easily corrected arises, such as strangulation or existence of an incarceration; (3) failure to retain the hernia by a correctly fitting truss occurs; (4) parental neglect persists, and (5) parents insist on the operation.

We do the Bassini operation on all infants, using one and never more than two sutures to close the posterior wall, catgut closure for the external oblique and horsehair for the skin. Our skin incisions rarely exceed 1¼ inches.

How shall we dress these? Not with a fluff and spika; but with a thin pad of gauze, the length of the wound, over which we place a gutta-percha covering which is sealed to the skin by collodion. After drying, in male babies, we use an apron or watershed of the same material, sealed to the pubes and dropped over the penis, so that the urine is diverted from the wound. Covers must not rest on the dressing. Infants are put in the regular hospital bed, which is surrounded and covered by one or more layers of sheeting, dropped to the floor, thus cutting off the under draft of air. The bed may be heated with hot-water bags hung as radiators. The child wears stockings and a shirt which does not extend below the navel, thus avoiding wetting and contact with the dressing.

The patients not operated on are treated by the use of trusses. Our results are satisfactory and there are few returns. We use the cross-body truss for all single and Hood's truss for double hernias. The material should be hard rubber, the strap excepted. When straps are wet they should be changed. The truss should be washed and dried once or twice daily and oftener if soiled; it will then give off no odors. The skin should be kept hard by hand friction and dusting powder. The truss must be worn day and night for curative purposes. It must fit properly; fit it yourself. It must not make pressure or cause friction sores; it must retain the hernia under all conditions.

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GENERAL ANESTHESIA IN THE SURGERY OF CHILDHOOD

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A presentation of the subject of general anesthesia in children has become an annual occurrence of the Section on Diseases of Children of the American Medical Association, and should be a valuable one if it does nothing more than recall to mind the constantly active factor that narcosis constitutes in the morbidity and mortality of childhood's various surgical procedures. In addition to thus refreshing the hearer's minds, it adds from time to time record of the advances that have been made in the technic and practicability of the art, and acts as an incentive to further investigation and study.

The specialist in anesthesia, as in every other branch of surgery, brings forth that which is ultrascientific, often complex, not that he so far lacks in common sense as to expect every practitioner to adopt it, but that, having the ideal in mind, the general standard may be elevated.

Several important physiologic differences between adult and child bear on the subject of general anesthesia. The pediatrist knows them well, but he may not connect them practically with the question of surgical procedure and narcotics. The physical inferiority of evolutionary childhood in terms of resistance makes the young subject especially sensitive to the toxemia of so grave an interference with nature as the administration of a chemical poison to produce loss of consciousness. Despite the presence of what might be considered as

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healthy life functions, there is in children a marked absence of that acquired immunity which adults attain through contact with their environment. In this light the child has a small physiologic factor of safety.

The proportion of heat-radiating surface to body-weight is much greater than in the adult; the heat-regulating nervous mechanism has not developed to the high plane of efficiency found later in life. Sudden or great loss of heat becomes relatively a much more serious factor. The total volume of blood in the circulatory system of the child is small, so that comparatively minor losses become relatively more serious and dangerous. The general circulatory efficiency and blood-pressure depend more completely on cardiac action, and less on a well-developed peripheral propulsive factor than in the muscular adult, so that slight interference with the cardiac action draws immediately on the margin of safety of the child.

The nasopharynx is distinctly smaller in relation to extraordinary respiratory needs[1]; its sensitive lining mucous membranes are easily stimulated to abundant secretion of mucus and its musculature to spasm, particularly about the glottis (recall the glottic occlusion of catarrhal croup and laryngismus stridulus). In the presence of lymphoid hyperplasia these obstructive and exudative factors are multiplied a hundredfold.

In children poorly understood dyscrasias exist, such as the condition called status lymphaticus, the thymus persists, and mechanically, as well as by its hypersecretion, makes for lessened resistance to the toxemia of anesthesia.

When serious accidents do occur in general anesthesia, in the majority of instances it is directly or indirectly the result of carelessness, the confidence of inexperience, or where familiarity has bred contempt and neglect.

It occurs in every discussion of the matter, as it does in literature, that many men see no sense or need for

1. Braislin, W. C.: Tr. Am. Otol. Soc., 1909, xi, part 2.

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the general warning against chloroform, and especially not in the case of children. Reports such as that of Grayson[2] of three thousand eight hundred chloroform anesthesias in nose and throat work without a death or serious collapse, prompt the promiscuous selection of this drug for such work without giving due weight, in considering the favorableness of the report, to the individual care and expertness which allowed the successful results reported. We all have considerable ego and easily conclude that we can do as well.

Chloroform has special advantages when used for children, and particularly in nose and throat work. These advantages prompt its selection by many, even in the face of its relatively great toxicity: 1. It is comparatively safe in the warmer climates. 2. It causes little, if any, increase of the nasopharyngeal mucus in cases in which such mucus dangerously adds to the respiratory obstruction which the operative manipulation inevitably produces. 3. It maintains an anesthesia for a longer period of time after the removal of the cone than does any other agent we employ—this is an advantage to the surgeon. 4. It is easy to carry and practical to administer. Despite all these advantages, however, the well-known and repeatedly demonstrated toxicity of the drug compared with others, makes it an agent to be avoided, and particularly so by the very ones who use it most, namely, the inexperienced, occasional users.

ERRORS OF TECHNIC THAT HAVE CONTRIBUTED TO AND CAUSED HIGH MORTALITY AND MORBIDITY, ESPECIALLY IN ADENOID AND TONSIL REMOVAL

Passing with mere mention the general necessity for minimum dosage of anesthesia and extreme care of administration with children, one comes to an analysis of the technical errors which cause damage or fatality in the usual anesthesia.

2. Grayson: Laryngoscope, 1913, xxiii, 61.

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In the case of chloroform, its special toxicity has been mentioned, but the error of administration which renders the toxicity active in the hands of the inexperienced has most often, according to my observations, been the continuous dropping, if not pouring on the mask, of the chloroform during the period of apnea, caused by a half voluntary holding of the breath, a spasm of the glottis, or an obstruction in the pharynx from blood and mucus. During this period of apnea the chloroform is collecting in large concentrated dose under and around the mask, lying in wait, so to speak, for the period of hyperpnea which will succeed the apnea, so that with this period of hyperpnea or deep, exaggerated breathing, the toxically concentrated and large volume of drug is sucked into the lungs with resultant paralysis of the central respiratory mechanism. Its threshold for stimulation being so lowered by the overdose of drug that the normal stimuli fail to maintain their influence, and death occurs.

In the case of ether, with its relatively slight toxic effect on the vital nervous mechanisms, the local irritant influence becomes active in producing an exudate of mucus or spasm of the glottis, the which, being greatly exaggerated in cases of lymphoid hyperplasia, becomes, in conjunction with the operative manipulations, a serious source of obstruction to breathing. This obstruction to breathing produces a marked degree of asphyxia in the subject for adenoid or tonsil removal, and at the same time prevents the further inhalation of the anesthetic. Recovery from the anesthetic prompts the anesthetist to apply large, concentrated dosage in order to subdue the patient and in virtue of this irritating dose the state of mucus exudation and pharyngeal spasm is again instituted. Thus is established a vicious cycle of too superficial anesthesia on the one hand, with obstructed breathing and damaging asphyxia on the other. Obstructive apnea alternates with overconcentration and overdosage, and gives an anesthesia than

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which there could be no more perfect for the inconvenience of the surgeon or more detrimental to the child.

At first glance this condition might seem to apply to cases in which surgery of the throat alone is in question, but the observation of any intelligent anesthetist or surgeon will demonstrate its existence as a cause of trouble in many anesthesias in children. The use of the drop technic in children on an open mask or covered with towels focuses every element of the danger that lies in its abstraction of heat or refrigeration on a subject the least able to withstand bronchial exposure or loss of bodily heat. The virtue of its comparative safety is swallowed up in the risk of its refrigeration. The only excuse for the use of any direct application of the liquid ether to the subject's airway is ignorance on the part of the anesthetist or sacrifice of patient's welfare to the convenience of the surgeon.

If there be any truth in Crile's observations on toxic nerve assault or anoci-association, we have good theoretical reasons to add to the frequent practical demonstration, that the extreme fear which accompanies many inductions of anesthesia does real damage to the subject's central nervous system and may have considerable influence in instances in which sudden anesthetic collapse occurs. In some cases a child may be anesthetized while asleep, but more often some speedy, sure technic must be adopted to shorten the seconds of agony for parent and subject. For this purpose no doubt nitrous oxid, properly administered, is superior, all things considered, the exception being in very young infants in whom the few drops of chloroform necessary to produce the desired result are less to be feared than the gas. Perfume, or 25 per cent. essence of orange, on the mask, prior to the chloroform, has its advantages.

The preponderance of narcoses in operations for adenoids and tonsils in children and the many difficulties encountered by anesthetists and surgeons in securing

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satisfactory anesthesia, make its consideration a factor of some consequence. Recently, French[3] has reported some investigations on anesthesias in throat work in children and during the use of the upright position has sought to attain the most satisfactory narcosis with the gas-ether sequence, Dawbarn's sequestration anemia, and an oxygen vapor of ether administered through a mouth tube during the operation and at intervals. The work of the operator is of the slow, painstaking kind, and thoroughness is his watchword, so that the anesthesia is of importance. The nitrous oxid initiation is found wanting in the claimed difficulty of the transition from gas anesthesia to ether. The oxygen ether vapor during the operation is complained of as probably augmenting the bleeding. Nasal insufflation is suspected of damaging the accessory sinuses of the nose.

In opposition to these views I believe that a nitrous oxid initiation is of sufficient value to warrant the acquisition of the skill necessary for the smooth transition from gas to ether vapor, and that such skill can be easily acquired. Also that the marked practical and theoretical value of nasal insufflation through tubes introduced well into the pharynx prompts the selection of this technic and that insufflation so used offers little, if any, chance of damage to the sinuses of the nose. The vapor-delivering mouth-gag, the oral tube, and the nasal tube deliver the anesthetic vapor outside of the area of obstruction to breathing in adenoid and tonsil work and thus do not overcome the respiratory obstructive difficulties of this anesthesia. Where tubes are inserted well below the soft palate, positive pressure within the nasal cavity and damage to the sinuses are negligible factors. The anesthetic vapor is delivered well behind the obstructive difficulties in the upper pharynx and if a proper volume of air is insufflated, the excess in the form of outflow tends to force the blood

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and mucus forward in the mouth where sponges or suction pump can remove them.

There are two operations especially that are performed on children in which the operator and patient are so handicapped by the mechanical question of anesthesia that I am certain that many deaths result directly because of the anesthetic. These two procedures are for sarcoma of the maxillae, especially the superior, and cleft palate. In both operations long periods of asphyxia are produced by the mechanical manipulation within the subject's airways, and in sarcoma of the superior maxilla especially from the hemorrhage that is bound to occur even with ligation of the external carotid.

INTRATRACHEAL INSUFFLATION ANESTHESIA

Tracheal insufflation anesthesia in children is especially easy of performance and offers such perfect anesthesia without one moment's anxiety for asphyxia or inhalation of blood and mucus, that any surgeon who operates once with its aid certainly would never attack a similar operation without it. The blood and mucus are actually washed out of the nares by the return flow of air, and the constant pink color of capillary circulation testifies to the complete oxygenation; the surgeon's work is not interrupted by the intermittency of the narcosis and the subject does not run the gauntlet of dangers from improperly maintained, recklessly toxic anesthesia.

In children the glottis is easily reached by the guiding index-finger of the left hand, the rubber catheter (18 to 22 size, with opening at the end and side) is easily introduced with forceps shown. The tube is then fastened to the angle of the mouth by a suture passed through the cheek and tube, and the anesthesia carried on by even so simple an arrangement as the vapor bottles shown in Figure 1.

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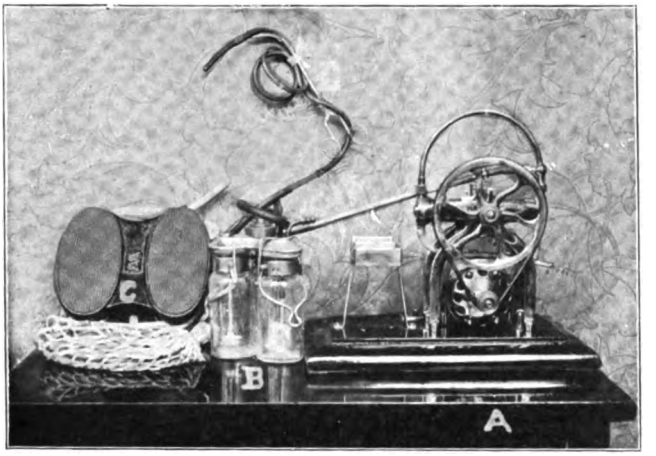

Fig. 1.—Vaporizing bottle set up for tracheal insufflation. *A*, electric motor pump; *C*, foot-pump; *D*, tracheal tube and forceps for introducing; *E*, third bottle of vaporizer used as mercury safety valve.

Fig. 2.—Nitrous oxid-ether vapor sequence apparatus. A, vaporizing bottles connected with bag *B* at *F*; *C*, foot-pump; *D*, Gwathney-Woolsey gas mask; *E*, gas tank connected with bag at *F*.

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ANESTHETIC TECHNIC ADOPTED BY THE AUTHOR

Sedative medication in the form of morphin products, etc., as a preliminary to anesthesia in children comes under the general head of contra-indications in the extremes of life, so that seldom, if ever, is it used.

Initiation of the narcosis is accomplished in as speedy and certain a manner as is consistent with safety, by the selection of one of two methods, viz.: Nitrous oxid gas or the essence of orange (25 per cent.) on a mask, followed by chloroform in carefully administered, intelligently observed dosage. These are followed by ether, in some way vaporized at a distance from the little patient's airways.

The gas-ether sequence is to be preferred if handled in a proper manner, and to be condemned if not so handled. The chloroform-ether sequence is safer than a poorly administered gas-ether. The technic which I use is the following:

The Gwathmey vaporizing bottles (A, Fig. 2) are properly filled with ether and water; 1 dram of essence of orange (25 per cent.) is placed in the water bottles. The foot-pump of the type shown (C, Fig. 2) is attached to bottles and from these to the lower end of the small breathing bag (B) and at this same point is attached a tube from a small tank of nitrous oxid. The bag is filled three-quarters full of gas; the mask (D) is placed quickly over the little patient's face, and in fifteen seconds unconsciousness ensues. After two breaths of gas have been taken, the foot-pump forces the weakest of ether vapors from the bottles into the bag where, like the gas, it is rebreathed, only a slight expiratory escape being allowed. This continues until deep ether narcosis ensues, at which time the breathing bag and mask are thrown aside and either a small vapor delivering Yankhaur mask is applied and connected with the bottles, or nasal tubes are inserted to the deep pharyngeal space. In either case, anesthesia by a non-refrigerating, minimum-dosed technic is continued. In ordinary anesthesia, that is, where access to the mouth or face is not needed, a towel or two, thoroughly covering the mask and confining the tidal air, gives the subject the benefit of considerable rebreathing with its carbon dioxid beneficence.

The ether indicator on the vaporizer rarely, if ever, goes above half way between ether and air. Instead of the foot-

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pump a stream from the oxygen tank may be used to carry over what ether vapor is needed, especially in those grave risks which test the skill of every surgical procedure.

The vapor method of administering anesthesia, including as it does such procedures as the pharyngeal insufflation recommended a year ago at Atlantic City by Dr. Pinneo, of Newark, N. J., and the tracheal insufflation for special surgical work, is one extremely easy of application, and because of its many virtues should be more generally adopted. An ordinary cautery bulb attached to a wash bottle of ether is superior to a drop technic on an open refrigerating mask and can be arranged by anyone without noticeable expense.

TECHNIC FOR ADENOID AND TONSIL WORK

As in any case initiation of the anesthetic is accomplished with the nitrous oxid-ether sequence, given as described above, and at the point where the breathing bag is thrown aside one or two soft rubber catheters are introduced through the nose and into the pharynx a distance equal to that from the external auditory meatus to the nares so that their ends rest just above the glottis, ether is thus insufflated to a point behind the obstruction of blood, instruments and fauces, and anesthesia is maintained by means of the foot-pump or the electric motor and pump (Fig. 1). The use of this latter means of air-supply furnishes coincidently one of the most valuable features of the technic, namely, a combined suction tube and tongue depressor. This suction tube, attached as it is to the air intake of the motor pump, and having an ordinary aspirating bottle between it and the pump, acts as a perfect aspirator of the throat contents during the operation, giving a clear field of vision to the operator and assisting materially the anesthesia by further freeing the airways of obstructing blood and mucus.

The vapor-delivery tube attached to a mouth-gag fails fully to maintain the narcosis because it delivers its anesthetic outside the obstructed area, and having the anesthetic vapor as strong as possible or in as large quantity as can be produced, avails nothing, inasmuch as the throat is obstructed at the fauces.

Nitrous oxid and oxygen as an anesthetic for adenoid and tonsil removal is not generally satisfactory, for the

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reason that it requires cumbersome apparatus to supply the large volume of gases needed in rendering them effectual through the nose; it produces a degree of cerebral congestion very productive of hemorrhage, and operator and anesthetist must be not only trained to work together, but in sympathy with each other's efforts and shortcomings.

RESPIRATORY RESUSCITATIVE MEASURES IN ANESTHETIC COLLAPSE

The very great majority of anesthetic deaths in both adults and children are of respiratory origin primarily, and in such accidents our efforts toward revival have been in no minior degree trivial. Artificial respiration as performed by the old recognized Sylvester or Shaefer methods has served well in some comparatively few cases, but its limitations are many. Oxygen, applied as it ordinarily is, by means of a tube placed in the nose, a funnel over the mouth, or similar procedure, almost provokes a smile when one realizes the state of the glottis, respiratory center and lungs when such collapse has occurred.

During a faulty narcosis with varying degrees of obstructed breathing, often progressively worse as the anesthesia proceeds, there is a constant negative pressure created within the chest by the violent muscular effort to obtain air through the obstructed throat, and when this continued asphyxia finally ends in respiratory collapse the epiglottis is so firmly sucked down against the vocal cords that its elevation by the finger is like pulling a cork out of a bottle; holding the jaw forward, even traction on the tongue under these circumstances is absolutely ineffectual in raising it, so that the ordinary artificial respiration with oxygen delivered outside the obstruction (in the mouth, for example) is next to useless. This condition of glottis and epiglottis was noted in utilizing the endotracheal tube for resuscitation of patients whom intern-anesthetists in hospitals had paralyzed, and in the presence of such physical conditions I believe that tracheal intubation offers the best means of

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quickly instituting artificial respiration in anesthetic collapse.

Another interesting phase of endotracheal artificial respiration has been developed in newly-born infants of atelectatic disposition where breathing has failed to materialize under the ordinary gymnastic manipulations. Any pediatrist who has used the O'Dwyer tubes can insert a small rubber, stiletted catheter into the glottis of a child without even the difficulty attending ordinary intubation, and though a mercury safety-valve between the oxygen tank and lungs is a primary requisite, emergency occasions could be met by a careful manipulation of the oxygen stream through the ordinary water bottle or a foot pump. Meltzer's recently described respiration valve effectually interrupts the stream of insufflated air or oxygen. This procedure in anesthetic collapse, sedative drug or gas poisoning, and post-partum atelectasis is a distinct advance in the means of resuscitating respiratory failure.

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PRECOCIOUS MENSTRUATION

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The term "precocious menstruation" should be limited to cases of more or less periodical menstrual discharge occurring in children before its usual appearance at the age of puberty (before the age of 9 or 10 years—Schauta), and should not include cases of hemorrhage from the vagina occurring within a few days after birth and not recurring subsequently.

Precocious menstruation is usually accompanied by signs of precocious maturity, i. e., "the premature development of the whole organism concurrently with menstruation and ovulation" (Morse[1]); or, in other words, "precocious menstruation with an early appearance of the external manifestations of puberty" (Croom[2]). These cases are by far the most interesting, and my case is of that type.

REPORT OF CASE

History.—Baby X., aged at time of examination 2 years (January, 1913). Adopted shortly after birth, at which time a Wassermann proved negative. History of parents unobtainable except that father was a Jew, mother an American. Labor normal. Weight at birth about 6½ pounds.

Bottle-fed, with modified milk. Weight at 1 month, 7 pounds; 2 months, 8¼ pounds; 3 months, 9⅜ pounds. At this time there was a severe gastro-intestinal disturbance with temporary intolerance for cow's milk. A wet-nurse was employed for several weeks, and then a weak milk modification. Weight at 4 months, 10¾ pounds; 5 months, 12

1. Morse, John Lovett: Precocious Maturity. Arch. Pediat., 1897, xiv, 241.
2. Croom, J. Halliday: Premature Menstruation. Allbutt's System of Gynecology, 1906.

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pounds; 6 months, 13⅞ pounds; 7 months, 16⅛ pounds; 8 months, 17½ pounds; 9 months, 21⅜ pounds.

The first tooth erupted at 10 months, at which time the menstrual flow first appeared, lasting three days. The discharge was of bright red blood from the vagina, with slight menstrual odor. At this time the child seemed rather large for its age, but no other signs of precocious maturity were noticed. Menstruation recurred at intervals of from one to three months, most frequently at intervals of six weeks. The child menstruated eight times in fourteen months. For a few days before periods a slight leukorrhea was noticed; the child was rather cross and acted as if in pain. She walked at 17 months after braces had been applied to support the limbs.

First Examination.—Large, well-developed, healthy looking child. Skin dark and hair very curly (Jewish type). Mentally bright for her age. Chest and abdominal examination negative as regards internal organs. Weight 38 pounds; height 37 inches; 17 teeth; circumference of head 19 inches; chest below breasts, 21 inches; across breasts, 22½ inches; abdomen at navel, 22 inches; breasts very prominent, about size of half an orange; nipples well developed, with slight areola; a few hairs around nipples; hair quite noticeable in the axillae, and pubic hair very noticeable; mons veneris and labia very prominent; hymen well developed, the opening admitting the tip only of the little finger, preventing a digital examination of pelvic organs. Rectal examination objected to, and photograph not obtainable.

Second Examination.—Age 28½ months. The child had menstruated four times in four and one-half months. Menstrual flow seemed to be increasing both in quantity and duration, and the peculiar menstrual odor was very marked. The child must wear napkins during a period of about a week. Leukorrhea was increased, being present most of the time. Weight 41½ pounds; height 39 inches; 18 teeth; circumference of head, 19 inches; chest below breasts, 21¼ inches; across breasts, 23 inches; abdomen at navel, 22 inches; pelvis, 23 inches. The breasts were noticeably more prominent, as also the growth of hair under the arms and about the external genitalia.

OTHER CASES

Lenz[3] reports in tabulated form 130 cases of precocious menstruation, dating back over 100 years. In addition to the case just reported I have found the following references to cases apparently not included by Lenz.

3. Lenz, J.: Vorzeitige Menstruation, Geschlechtsriefe und Entwicklung, Arch. f. Gynäk., 1913, xcix, 67.

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Raffaelli[4] reports a case, D. E., aged 4½ years, menstruating for several months, lasting about eight days, preceded by abdominal pains. Height, 1 meter, 10 cm., weight, 19 kg., and average build of a 6-year-old girl. No syphilis, alcoholism or other hereditary disease. Hair in axillae, mammary areola very noticeable, but no enlargement of the mammary glands. Mons veneris prominent and covered with hair about 1 cm. long. Labia very much developed, resembling those of a girl 10 or 12 years old. Hymen well developed and the opening therein permitting introduction of tip of little finger. No evidence of pelvic tumors.

In his article Raffaelli refers to the bibliography on the subject in the work on Children's Diseases by Grancher and Comby. He also refers to a number of similar cases, most of which are included in Lenz's tabulation. The apparent exceptions are as follows:

Reim: The case of a girl 6 years old, who menstruated at 5 years; physical signs of puberty of a girl 13 or 14 years old. Abdominal examinations revealed presence of an ovarian cyst.

Woodruff[5] reports a case of a girl 6 years and 2 months old who menstruated at the age of 2 years. She was developed, apparently, like a girl of 12 to 14 years.

Barband and Lefevre[6] report a case of Puech of a girl who menstruated at the age of 4 years. When 8 years old she had sexual intercourse and miscarried at three months.

Dotti[7] at the meeting of the Tuscan Pediatric Society in 1908 presented three cases of 8, 3 and 2 years. These three cases were the only cases noticed out of 6,000 patients during a period of two years at the Pediatric

4. Raffaelli, G.: Un caso di mestruazione precoce, Estr, dall' Osp. d. Bambini di Milano, 1912, No. 1; Abstr. Brit. Jour. Child. Dis., 1912, ix, 235.

5. Woodruff, E. G.: Case of Unusually Early Menstruation. Med. Rec., 1896, xlix, 338.

6. Barband and Lefevre: Essai sur la puberte chez la femme, Paris, 1897; Quoted by Raffaelli (see note 4).

7. Dotti: Riv. di clin. Pediat., Maggio, 1908; Quoted by Raffaelli (Note 4).

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Clinic, Florence, Italy. In one of the three cases the important factor was a suspicion of hereditary syphilis (premature birth, mother advanced in years). In all three the general body development was above the average, with psychic deficiency. One of the girls had a marked development of the thyroid.

In the discussion of Raffaelli's case, Professor Mya cited a case of precocious puberty seen by him in a girl 4 years old, and Dr. Funaro, a case of precocious menstruation that he had seen at the Charité in Berlin, in a girl of 5 years. It is possible that this is the same case seen by me at the orthopedic clinic of Professor Hoffa in 1908, at which time the following notation was made:

Girl nearly 5 years old has menstruated almost regularly since 7 months old. Precocious physical development first noticed when about 1 year old, and from that time has grown very rapidly. Breast, pelvis, external genitalia and body growth that of a girl 10 or 12 years old. Hair in axillae and pubic hair very noticeable. Intelligence about normal for age. Comes to clinic for left-sided coxitis. Cast applied.

Raffaelli observes that "Many of the cases present pathological conditions such as tumors, abnormal development of the thyroid, suspicion of hereditary syphilis, rickets, obesity, hydrocephalus, adenoids, psychic deficiency, etc." He further observes that "According to some authorities, beside the climate, the altitude, the proximity of the sea, country or city life, the social condition, the color of the hair, the mode of living, education and race would seem to have a certain influence on menstruation; in fact, it is proved that the Jewesses menstruate before many other women (the Slavs for instance) who live in the same climate."

The fact that these cases are a source of embarrassment and worry to the parents, as well as to the children themselves when they become old enough to appreciate their condition, ought to be reason enough to bring up the question whether anything can be done to con-

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trol the condition. Glynn[8] refers to two remarkable instances of sexual development in children with a tumor of the ovary (Lucas[9]) and of the testicle, respectively, in whom "The former childlike appearance returned after the removal of the tumor." He also refers to seventeen examples of sex abnormalities in children associated with adrenal hypernephroma verified post mortem, the abnormality in most of the cases being of the type of precocious maturity. He further speaks of the relationship of the pituitary gland to the adrenal gland and sex characters; also of the pineal gland and sex characters. Thus he says "tumors in the pineal region, though rare, are occasionally, like those of the adrenal, associated with changes in sex characters, particularly true precocious sexual development.

Congenital hypertrophy of the pituitary gland is frequently associated with giantism and congenital hypo-secretion with infantilism (Glenn[10]). There is probably also some relationship between the ovaries and the thyroid gland.

Since these cases of precocious menstruation and precocious maturity are, undoubtedly abnormal and probably pathological, I would like to leave with you for discussion and for future thought the question whether it is permissible to administer experimentally some remedy, perhaps some glandular substance, to counteract this tendency to precocious development.

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8. Glynn, E. E.: The Adrenal Cortex, Its Rests and Tumors: Its Relation to Other Ductless Glands, and Especially to Sex, Quart. Jour. Med., 1911-1912, v, 186.

9. Lucas, C. R.: Tumor of the Right Ovary in a Child Aged 7 Years Associated with Precocious Puberty, Tr. Clinical Soc., London, 1887-1888, xxi, 224.

10. Glenn, T. H.: Internal Secretions, Denver Med. Times, May, 1913, p. 533.

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THE HOSPITAL MANAGEMENT OF CONTAGIOUS DISEASES

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It has long been believed, and still is, that the so-called "contagious diseases," comprising such diseases as scarlet fever, diphtheria, whooping-cough, measles, small-pox, chicken-pox, mumps and rubella, are to a large extent contracted by aerial transmission. Much evidence against this view has accumulated during the last few years and one purpose of this paper is to discuss this evidence.

It is only the practical experience of the hospitals of England and France and the Providence City Hospital that will be presented to show the importance of contact infection and the relatively small factor, if any, of aerial transmission. The paper is not based on laboratory findings.

The French were the first to doubt the theory of airborne disease and the importance of controlling contact infection. To Grancher of Paris belongs the credit of being one of the first to put these ideas into practice. He isolated contagious diseases in the wards of a general hospital. Wire screens were placed about the beds to indicate that certain precautions were to be taken in handling these patients. These precautions were based on the method of strict asepsis. From 1890 to 1900, among the 6,451 patients admitted to Grancher's wards, diphtheria was introduced forty-three times and only once did the disease develop in the ward; scarlet fever

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was introduced nineteen times and seven cases developed. Less success was obtained with measles, although infections were reduced two-thirds. The evidence was, however, conclusive enough to convince Dr. Grancher that even measles was not spread by air.

Out of this simple but important beginning have developed various methods of construction for segregating the various diseases. Aseptic nursing is employed, whatever the type of separation. Some disregard air separation; others aim to interrupt, partially or wholly, air exchange between compartments occupied by patients. All plan to establish physical separation and to prevent contact. Out of Grancher's method developed the barrier system which is largely used in England. This is the method of isolating patients in a common ward. The beds are generally placed on centers 12 feet apart. In the beginning, the beds were surrounded by sheets kept wet with bichlorid of mercury. At present the wet sheets have been dispensed with and the bed is marked in some way. In one hospital, two uprights are set on the floor, one on each side of the foot of the bed, and a cord of colored tape is stretched between. This serves merely to indicate to nurses that precautions are to be taken.

Another method is the so-called cubicle system. The cubicles are small rooms with partitions more or less complete. In many instances the partitions are about 7 feet high. They are of silicon-plaster, glass, or a combination of the two. The rooms are arranged either on one side or on both sides of a common corridor, or sometimes in England, old wards have been cut up into separate compartments by partitions which do not reach the ceiling, all compartments being ventilated from the common air-space above. The cellular block system consists of rooms back to back with glass partitions; access to the rooms is from verandas.

Permit me to report some of the results in English hospitals where medical asepsis has been tried since

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1907. In the Plaistow Hospital, Dr. Biernacki has been using the barrier system extensively, as well as the cellular block system. Dr. Biernacki writes that he would isolate the following diseases with the barrier system:

1. Diphtheria.
2. Whooping-cough.
3. Mumps.
4. Rubella.
5. Typhoid fever.
6. Septic infections.
7. Ringworm, except when not additional to scarlet fever.

He does not attempt to isolate measles or chicken-pox, and hesitates to do so with scarlet fever. In his cellular block system all of the before-mentioned diseases are treated as well as typhus. In the block there has been no cross-infections for three years. He quotes no statistics relative to his barier work, as he has not reached a final decision as yet.

Dr. F. Foord Caiger, medical superintendent of the SouthWestern Hospital, London, reports on eighteen months from Jan. 1, 1907, to June 30, 1908, on the cubicle system. The partitions are 7 feet high, the lower half being granite silicon-plaster and the upper half glass in a metal frame. Seven hundred and four patients were placed in these cubicles, of whom 289 were scarlet fever convalescents who were placed in them two days and nights before their discharge. The remaining 415 were isolated in them for the following reasons:

1. The original diagnosis was uncertain.

2. The disease, though recognized, was one that called for separate isolation.

3. The patient had been exposed to another disease prior to admission and therefore was possibly incubating a second disease.

4. The patient was suffering from more than one infectious disease.

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5. The disease was obviously of a non-infectious nature.

In addition to the 289 convalescent scarlet fever cases referred to above, the following diseases were placed in cubicles:

Scarlet fever, 163 cases, in the eruptive stage of the disease.
Rubella, 82 cases.
Measles, 22 cases.
Diphtheria, 31 cases.
Whooping-cough, 17 cases.

Of these 704 patients, twenty developed a second disease in the cubicles. Three of this number, however, were incubating the disease on admission and a fourth was infected by a ward maid who was suffering from diphtheria. Of the sixteen diseases arising in the cubicles, six were scarlet fever, six were chicken-pox, two were rubella, and two were measles. In two instances, one chicken-pox and one measles, there had been no recogized case of the same disease previously in the ward for a period of six weeks and two months respectively. Thus only fourteen persons apparently contracted another disease from patients in the cubicles. It will be seen that no case of whooping-cough or diphtheria developed. Dr. Caiger concludes that of all the before-mentioned diseases, chicken-pox is the only one which he has ceased to put into cubicles because of the six cases arising in the first six months of the period reported on. In his 1911 report he speaks as follows:

The incidence of secondary infectious disease among the 269 patients treated in cubicle wards was 1.8 per cent., whereas in general wards it was 7.6 per cent. In addition to these 269 patients, chicken-pox and measles being the only excluded, 177 scarlet fever convalescents were placed in one of the cubicle wards for the last two days and nights of their stay in the hospital. In all, 2,547 patients have passed through the cubicles during the five years they have been in use. As an extension of isolation provision they have proved of the utmost service.

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Dr. Thompson of the North-Eastern Hospital, London, reports on an experience of two years with the cubicle and box-room system. To the cubicles, 1,290 patients were admitted. The following cross infections have developed:

	Cases		Cases
Scarlet fever	5	Whooping-cough	1
Rubella	3	Chicken-pox	3
Measles	2	Diphtheria	2

His conclusions are rather conservative. During the last twelve months, he placed in the cubicles doubtful cases of scarlet fever and diphtheria and is averse to placing other infectious diseases in them.

To the box-rooms, 660 patients were admitted, suffering from the various infectious diseases except chicken-pox. The following cross-infections developed:

	Cases
Scarlet fever	6
Rubella	3
Measles	2
Whooping-cough	1
Chicken-pox	3

In his 1910 report, Dr. Goodall, medical superintendent of the Eastern Hospital, London, describes the conversion of a long ward, 106 by 28 feet, into twenty cubicles with glazed partitions. Ten rooms open from both sides on a common corridor. These cubicles were opened for patients Sept. 6, 1910. Up to March 25, 1911, 142 patients passed through this ward. Of these, 110 suffered from an infectious disease, while thirty-two did not. The following diseases were included:

	Cases		Cases
Scarlet fever	16	and chicken-pox and scabies	1
and diphtheria	2	Diphtheria	5
and measles	5	and measles	5
and chicken-pox	9	and chicken-pox	3
and rubella	2	and rubella	1
and whooping-cough	1	and whooping-cough	2
and diphtheria and measles	1	and mumps	2

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	Cases
and measles and chicken-pox	1
and measles and rubella	1
Measles	25
and chicken-pox	1
and rubella	1
Chicken-pox	3
Rubella	13
Mumps	1
Erysipelas	1
Influenza	1
Enteric fever	1
Lobar pneumonia	4
Scabies	1
Diseases non-infectious	32
Total	142

Patients were not selected as to the stage of the disease or as to position in the ward. One child contracted chicken-pox during the six months' period.

Dr. C. Rundle of the City Hospital, Liverpool, reports that during 1910 and 1911, 668 cases were treated by the barrier system. These cases included all contagious diseases, not excepting measles and chicken-pox. During these two years, only two patients developed a secondary disease, both contracting scarlet fever. Other hospitals in England which are employing aseptic nursing are situated at Manchester and Walthamstow.

The earliest and parent hospital, the Pasteur in Paris, is perhaps one of the most successful. Small-pox is admitted to this hospital, which is done in no other hospital so far as I am aware. I know of only one small-pox cross-infection. Two other Paris hospitals, the Herold and the Hôpital des Enfants Malades, employ aseptic methods, but I do not know their results.

I shall now offer for consideration an experience in the Providence City Hospital of three years, from March 1, 1910, to March 1, 1913, with aseptic nursing of infectious diseases. The special construction of this hospital and introduction of aseptic nursing in this country is the work of Dr. Charles V. Chapin, superintendent of health of the city of Providence.

Patients are accommodated in three two-story buildings' situated parallel, one each for diphtheria and scarlet fever, and one an isolation building, so-called, for all other infectious and mixed cases. Each floor of the diphtheria and Scarlet fever buildings is divided

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into a ward of twelve beds and seven or eight rooms arranged on either side of a central corridor. The side rooms vary in size, containing from one to three beds. The ground floor has also an admitting room, connecting with a doctors' gown and wash room, and a patients' bath. The large number of small rooms furnishes the opportunity for isolating mixed or doubtful cases and for detaining new cases a certain period of time before they are allowed with convalescents. Each serving kitchen is provided with a utensil sterilizer for sterilizing dishes, etc.

Each of the rooms, except the large ward, is provided with a lavatory with forearm levers and without plugs, hooks for gowns, a bar on which to hang individual towels and a small shelf for the patient's thermometer, etc. The usual nursing utensils are kept on a bedside table. All the paint has an enamel finish so that it can be easily washed.

On the first floor of the isolation building are twelve single rooms arranged on either side of a central corridor. Ten of them are arranged directly opposite so that the doors open opposite to each other. Beside each door and opening into the same room, is a full-sized window which permits of better observation of patients by the nurses and keeps the patients better contented by permitting them to see their neighbors across the hall. The partitions rise to the ceiling and are of studding, lathing and plaster. Each room is furnished the same as those just described. This floor also has its serving kitchen, toilet, bath and operating-room. In the serving kitchen is a large utensil sterilizer for sterilizing dishes, medicine glasses, etc., which come from the patients.

The construction of the second floor of the isolation building is similar except that the rooms contain from one to three beds, making it possible to accommodate in the same room several patients isolated for the same purpose. The doors to all the rooms in the building

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are always left open unless there is some unusual reason for closing them.

The dormitory for nurses is in the administration building; that for female help is in the service building and for male help over the power house and laundry.

The administration of the hospital has been worked out along the line of avoiding contact infection. It has been our aim to avoid cross-infection and infection of employees by strict asepsis. Every new nurse and employee who has to go into the wards is thoroughly impressed with the idea that if he gets sick it is most likely his own fault and probably due to putting his fingers or something else contaminated in the ward into his mouth; employees understand that if any cross-infection develops they are liable to investigation as to the care they exercise in their work. Every employee has a culture taken on beginning work and before going into the wards. Everyone must be vaccinated before or soon after entering on his work. No prophylactic antitoxin is administered either to employees or to patients.

I shall not attempt to present all the details of our technic, but shall outline the more important; first, those relating to the general administration, and second, the more direct care of the patient.

The resident physicians have their quarters in the administration building and all eat in the same dining-room. While working in the wards, white duck suits are worn, and in each ward a gown is put on if it is necessary to make examinations and thus come in contact with the bedding and patients. On leaving the ward, the gown is removed in the gown and wash-room and the hands are carefully washed with soap, water and brush in the special basin to which I have already referred. Great care is exercised when not wearing a gown to avoid allowing any part of the clothing to touch the bed, bedding or anything else. Physicians wear the usual hospital uniform and in addition a short-sleeved jacket to avoid sleeve infection.

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The nurses all sleep in the same home and eat in the same dining-room. When off duty, they are allowed to leave the hospital as freely as from any general hospital. When the nurse goes on duty, she goes to the dressing-room for that ward where she has two metal lockers, one for clean clothes and one for infected clothing. She puts on her ward clothes and goes to the ward. She changes her dress, cap, apron and bib only. When going off duty, she removes her infected clothing first, washes hands and face, and then puts on her uninfected uniform. The sleeves of the uniforms are either short or rolled up.

The maids and other help put on gowns when entering the wards. These gowns have short sleeves so that the sleeves will not be contaminated. Otherwise employees observe the same care as do the nurses. The ward help eat in the same dining-room and sleep in the same quarters with all the other help, and they, too, are free to come and go when off duty. It is insisted that every case of illness among nurses and help be reported at once in order that no mild infection may be overlooked.

The same kitchen furnishes food to patients and employees. Food supplies are sent to ward kitchens in paper bags and paper trays, and these are destroyed. Such dishes as must pass between kitchens are boiled before leaving the ward and washed again in the main kitchen.

All hospital linen is washed in one laundry, the washing being done in ordinary washers.

The greatest source of infection is certainly the patient himself. If we are to minimize diseases arising in the hospital, we must have very careful regulations and insist on their obedience. Our method of procedure has been based on the contact infection idea and we have disregarded air transmission unless one chooses to insist that the coughing of a patient directly into one's face is air-borne infection.

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I will trace briefly the method of care from the time the ambulance goes to the house until the patient is ready to walk out of the hospital. The various infectious diseases, including tuberculosis, are brought to the hospital in the same ambulance or in a coupé. There is nothing peculiar about the construction of these vehicles, except that the interior can be freely washed with soap and water without damage. The attendant and the driver wear clean, washable coats on every ambulance trip. Careful inquiry is made as to the presence of other recent previous infection of the patient or of any one in the house. The patient is wrapped in blankets and brought to the hospital, where he is placed in the appropriate admitting-room. The driver and the attendant return to the stable, remove their coats and wash their hands. The infected blankets and coats are put in a hamper and later sent to the laundry. The interior of the coupé or ambulance which has come in contact with the patient is washed with soap and water.

The patient remains in the admitting-room until the admitting officer has made a physical examination. A culture nose and throat and vaginal smear are taken before admitting to the ward. Even though it may be an uncomplicated case belonging in that ward, every new patient is held in detention from five to seven days, and often longer. "Detention" means that the patient is placed in one of the rooms off the ward used for the purpose where aseptic precautions are observed. Should there be any doubt at all about the diagnosis, or exposure to or suffering from mixed infections, the patient is sent to the isolation building, or else is barriered by placing a red card on the bed.

Briefly, our technic in the care of detention and barriered case is as follows: The patients may occupy single rooms off the ward or be in detention separately or collectively in larger rooms. They are kept in bed and are not allowed toys or anything else which can be thrown from bed to bed. The hands of doctors and

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nurses are washed after treating the patient. Each patient is supplied with a thermometer, basin, pus-basin, bed-basin, bed-pan, etc., which are kept on a table beside the bed. There is a gown to be used when coming in direct contact with the patient. All dishes are boiled after using. Our object is to control in-going and out-going infection to or from new cases.

The construction of the isolation wards has been referred to. The furniture is small in amount, and, like all the ward furniture, easily cleaned. The isolation wards have proved the most valuable section of the hospital. In them we have not hesitated to treat any of the so-called contagious diseases, except small-pox, in any stage, as well as cases for observation only. We are obliged to treat cases of measles, chicken-pox, whooping-cough, rubella, mumps, etc., and it has been very convenient and, we believe, safe to use these single rooms, equipped as they are. It saves opening a large ward and furnishing a separate supply of nurses and maids for each.

We confine the infection to the rooms immediately occupied by patients. We believe that the corridors, serving kitchen, linen-room, bath and toilet are as safe and free from infection as those of any general hospital. The same nurses care for the patients on a single floor. The nurse washes her hands with bar soap in running water, using a brush, and dries them on an individual towel every time she touches the patient or anything in the room. After handling measles and chicken-pox, in addition to the washing, a one-minute immersion in an antiseptic solution is required. If the nurse comes in close contact with the patient she puts on a gown. Each patient is supplied with everything as described above. All dishes and nursing utensils coming from these patients are put directly into a sterilizer, if steam can be used, or 1:20 phenol (carbolic acid) solution is used for rubber and glass goods. Bed-pans and urinals are emptied into a hopper and put into a tub of 1:20 phenol

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solution, in which they are kept an hour or more before removal to the warming rack.

Small children who would play on the floor are kept in bed throughout their hospital residence. Larger children or adults are allowed up, but confined to their rooms, except when they are allowed outdoors, when they are also kept apart. The doors of the rooms stand open all the time, and this practice, together with the extra corridor window, allows patients to see each other and to talk back and forth, and they seem very contented.

When a patient is discharged, the linen in his room is sent to the laundry, the mattress and pillows to the steam sterilizer, and nursing utensils are sterilized. The bed, wash-basin, table, chair, floor and walls within easy reach are simply washed with soap and water. Preferably the room is aired for twenty-four hours, but if we need it we have not hesitated to put a new patient into it immediately. Rooms are never fumigated.

Every discharged patient receives a thorough soap and water bath including a shampoo. The clothing has been sterilized by steam, 10 pounds for thirty minutes, so far as possible. Those articles which steam would injure are treated with formaldehyd vapor in the same chamber, to which 1 or 2 pounds of steam has been added.

It will be noticed that we do not use any disinfectant solution for the hands, except after measles and chicken-pox. It has seemed to us that if we could make it convenient enough to wash the hands with soap and water and brush every time they became infected, it would be sufficient. There is no antiseptic solution solution which is efficient under immersion of less than one minute. An antiseptic solution is not necessary after washing, except perhaps in the case of measles and chicken-pox.

Time will not permit me to present the results of aseptic nursing after three years' experience in such

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detail as I should like to. I believe that the Providence City Hospital is the only hospital in this country which keeps an accurate record of all diseases contracted in the hospital by patients and help, and publishes it in annual reports. I sincerely wish that it were a general practice so that we might have facts on which to base comparisons of the results.

I shall first mention briefly the number of diseases contracted in the hospital, with incidence of infection, among help, scarlet fever patients and diphtheria patients; then take up in more detail the results of treatment in our isolation wards, which is the real test of aseptic nursing.

Among the seventeen physicians who have worked in the wards, the only disease contracted was diphtheria, three cases, 18 per cent. Among forty-seven graduate nurses, scarlet fever developed once, 2 per cent; diphtheria twice, 4 + per cent; rubella once, 2 per cent. Among 183 pupil nurses who work in the wards two months, scarlet fever occurred eleven times, 6 per cent.; diphtheria ten times, 5.4 per cent.; rubella once, 0.5 per cent. Among eleven nurse maids, no disease occurred. Among eighteen ward kitchen maids, one developed scarlet fever, 5.5 per cent., and one diphtheria, 5.5 per cent. One laundry maid developed scarlet fever and a night telephone operator diphtheria.

Diseases contracted among the 861 patients who have been admitted to the diphtheria ward are as follows:

Scarlet fever	2	Rubella	3
Diphtheria	1	Whooping-cough	3
Chicken-pox	18	Total	27

This constitutes an incidence of secondary infection of 3.1 per cent.

Diseases contracted among the 649 patients who have been admitted to the scarlet fever ward are as follows:

Diphtheria	3	Chicken-pox	13
Measles	4		—
Rubella	1	Total	21

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This constitutes an incidence of secondary infection of 3.2 per cent.

The diseases contracted among the patients in the isolation building have been confined to measles, chicken-pox and scarlet fever. Between March 1, 1910, and March 1, 1913, the period which this paper covers, there have been treated in this ward 842 patients. Of this number, 151 were there less than a week, and 691 more than a week. These 842 patients suffered from the following diseases:

Chicken-pox	76	Syphilis	1
Diphtheria	137	Tonsillitis	22
Diphtheria carrier	138	Tuberculosis pulmonary	16
Erysipelas	3	Vaginitis gonorrheal	8
Influenza	1	Variola	2
Measles	242	Vincent's angina	4
Mumps	11	Whooping-cough	128
Noma	6	Tinea tonsurans	1
Ophthalmia gonorrheal	2		
Rubella	37		1,066
Scarlet fever	231	Non-infectious diseases	88
		Total	1,154

The excess of diseases over the number of patients is accounted for by mixed cases.

The following diseases have been contracted: chicken-pox, 8 cases; measles, 25 cases; scarlet fever, 10 cases; a total of forty-three cases and an incidence of 5 per cent. Remember that the foregoing diseases were placed in the isolation rooms in any and all stages.

So far as I know no other hospital, except the Pasteur, has ever undertaken to care for such a variety of diseases by aseptic nursing. It is fair to conclude that the following diseases can safely be treated by the cubicle system: diphtheria, scarlet fever, whooping-cough, rubella, mumps, gonorrheal infections. There may be some question with relation to chicken-pox and measles. I believe that when our technic is better perfected, these also can be recommended as amenable to aseptic nursing. I believe that the cross-infections were due to faulty technic. The cross-infections have appeared mostly in

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outbreaks and we have had intedvals as long as fifteen months in the isolation wards without a secondary disease arising.

ABSTRACT OF DISCUSSION

DR. H. E. ROBERTSON, Minneapolis: I wish to ask one question in regard to the pupil-nurses. Does he place pupil-nurses in his isolation ward, or does he use trained nurses for this strict aseptic work?

DR. D. L. RICHARDSON, Providence, R. I.: For the first eighteen months of our work in this isolation ward we used pupils, but after one or two experiences we felt that it was not safe. If we had pupils for six months or a year we should deem it feasible, but we have them for only two months. It is not long enough to trust them to nurse cases of measles and chicken-pox and possibly scarlet fever. If they are in the ward for several months' preliminary training then they can be trusted. Our object is to develop an aseptic technic. The nurse is under strict supervision for a certain period of time because we must have faithful assistants. The work in fact depends entirely on the nurses. They are the ones who care for the patients, and it is necessary to enlist the cooperation of every one in the hospital to insure success. I am glad to say that in our work every one has imbibed the spirit of aseptic nursing and does not fear the diseases in any way, being careful to avoid any opportunity of contact.

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THE FEDERAL CHILDREN'S BUREAU—SOME ASPECTS OF THE PRESENT WORK

JULIA C. LATHROP
WASHINGTON, D. C.

The Children's Bureau is the result of efforts lasting for ten years to secure a provision by which the general government should create a special agency devoted to the interests of the children of the nation, with methods of investigation and publication analogous to those through which the various bureaus of the Department of Agriculture have vastly increased the productiveness of the soil and the welfare of farmers.

In these efforts many organizations and individuals joined, notably the National Child Labor Committee, various settlements and women's clubs, societies and associations engaged in the active work of caring for children and protecting the public health. So that the Bureau went into operation with a remarkable backing of public interest and good will.

The Children's Bureau was established by Act of Congress on April 9, 1912, although it did not go into operation until August 23 of the same year, when the appropriation for its maintenance became available. The statute defines its duties thus:

> The said bureau shall investigate and report to said department on all matters pertaining to the welfare of children and child life among all classes of our people, and shall especially investigate the questions of infant mortality, the birth-rate, orphanage, juvenile courts, desertion, dangerous occupations, accidents and diseases of children, employment, legislation affecting children in the several states and territories. . . . The chief of said bureau may from time to time publish the

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results of these investigations in such manner and to such extent as may be prescribed by the Secretary of Commerce and Labor (now Secretary of Labor).

For the discharge of these duties, the statute provides a staff of fifteen persons and an annual appropriation of about thirty thousand dollars.

It is apparent that this is a bureau of investigation and publicity, and although it is directed to investigate "all matters," that direction is to be interpreted with relation to other existing bureaus of the government, notably those of the Census, Education and Public Health, which have to do with certain aspects of the care of children and with which this bureau naturally cooperates.

It is obviously intended that the Children's Bureau shall occupy the field popularly described as that of "child welfare," which must be based on census enumerations and must touch on the social, economic, and moral aspects of both education and health.

If the work of this new bureau be of interest to this body, it is not because that work is medical, for it is not; nor because the staff of the bureau includes physicians, for it has none. It is rather because you have so enlarged the field of your professional interests by the splendid sweep of preventive medicine, to which nothing is alien, that you include this bureau.

INQUIRY INTO INFANT MORTALITY

The law itself mentions infant mortality as the first subject to be undertaken, and it is certainly reasonable that the government should consider first of all this subject—the wastage of human life at the beginning. If the census authorities are right in estimating that at least 300,000 babies, or one out of every eight born, died last year before any one of them had lived a twelvemonth—if, of all the funerals in America, one in every five is that of a child less than a year old—and if, as you tell us, at least half of these deaths are preventable

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by methods that you as sanitarians have ascertained and placed at our disposal—then the American public cannot disregard the call to action, nor can we ignore the larger implications of social welfare presented by the statement of Newsholme:

> **Infant mortality is the most sensitive index we possess of social welfare. If babies were well born and well cared for, their mortality would be negligible. The infant death-rate measures the intelligence, health, and right living of fathers and mothers, the standards of morals and sanitation of communities and governments, the efficiency of physicians, nurses, health officers and educators.**

Again, we are challenged by the report of the New York Special Public Health Commission, just issued, which shows for that state that, since 1902, the rural and village death-rate has slowly risen; that it began to exceed the urban death-rate in 1909; and that "since that date the divergence between the two, in favor of the urban death rate, has steadily increased."

The report of the commission continues: "The work of this commission may be said to be to ascertain how the rural and village death-rate can be made to follow the urban death-rate in its downward course."

It is safe to say that the improvement in urban health is in large measure due to the new attention paid to saving infant life. This is shown by the improvement in the infant death-rate of London, which in the last ten years has been reduced from 160 to 103; so that to-day the London infant death-rate is lower than that for all England, which is 105. It is startling indeed to find that the untouched sore of the health problem to-day is not in our congested cities, with all their acknowledged unwholesomeness of life, but in rural and village life, on whose wholesomeness we have confidently depended to reinforce our cities.

On the other hand, it is deeply significant that the most favorable record of infant mortality is that of New Zealand, where the figure was 68 in 1910—a country whose newness, equality of opportunity, wholesome

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climate, and high level of intelligence combine to give favoring conditions, but only such conditions as this country should also afford.

Partly because the limited appropriations forbade the bureau's undertaking extensive inquiries for the present, and partly because the great cities of this country have already under way many successful civic and volunteer activities for reducing infant mortailty—while the necessity for such work, being less obvious in smaller communities, is not yet thoroughly recognized—it was determined to undertake a series of studies into infant mortality in small industrial centers and rural communities as our first field work, and it is a preliminary statement regarding the method of this inquiry which I wish to submit to you.

METHOD OF CONDUCTING THE INQUIRY

Our inquiry does not assume to be a medical inquiry, nor even one based on the records of deaths. It is rather an inquiry into the social, industrial and economic conditions surrounding the children born in a given town within a given year, and is an effort to trace every child so born either through that year, or as much of that year as he survives.

It was necessary, in conducting an inquiry thus based on the birth records, to choose localities within the registration area. Johnstown, Pa., was selected because its size, situation in a registration state, and general industrial characteristics made it a suitable beginning.

The schedule follows the child through the first year of life in his family surroundings, and in addition embraces a survey of the reproductive history of the mother. It is necessarily intricate and perhaps annoying to the person who must answer the questions.

The inquiry was launched with some anxiety as to how the agents would be received. The field agents were, of course, necessarily women. The law states that "no official, or agent, or representative of said bureau

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shall, over the objection of the head of the family, enter any house used exclusively as a family residence." The instructions to the agents were, in addition to this, that they should enter no home without the good will of the woman at the head of the family, and for two reasons: first, because the humbler the home, the greater its need that the government should recognize its dignity; and second, because without the good will of the woman at the head of it, no schedule could be prepared which would be worth writing down. It was doubtless an aid in securing the cooperation of the mothers that the schedule was arranged so that the name and address appeared on a perforated slip which was removed in the mother's presence, thus assuring her that the schedule was returned to the office without a name on it and that the government only desired the facts, without any tinge of personality.

Fifteen hundred and fifty-eight schedules were secured. It is interesting to note that there were two refusals.

It is obvious that such an inquiry is absolutely democratic, since it must follow the history of every baby wherever born, if born within the given year. The agents found everywhere a cordial reception. Whether among the foreign-born women who must be visited with an interpreter, or in the American quarter of the town, the mothers responded to the request of the government to cooperate in an effort to learn how to safeguard their own and their neighbors' babies' lives. The State Board of Health sent for distribution in the foreign quarters circulars in various languages on the care of babies. The American mothers took our agents to task because the government had no instructions in English for them, complaining of the confusion of doctrine among physicians, nurses, neighbors and relatives.

LOCAL INTEREST CREATED

Such an inquiry should bring out an invaluable by-product of local attention, and this is shown in the

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present instance by the interest of the health department, the press, the women's clubs, and the clergy; by renewed public activity in such matters as the disposal of garbage; and by the request of the city that the Department of Agriculture make a thorough examination of the milk-supply. This examination is now under way.

It would hardly seem worth while to take up your time with a description of so slender an inquiry, were it not to be considered the beginning of a series which is to be carried on, securing comparable data in every instance. The report of each inquiry is to be made independently and published as promptly as it can be prepared. Later it is intended to collate all the material.

There are various aspects of the present work of the bureau which I will not discuss here, but I may briefly mention that the bureau has now in preparation a series of pamphlets on the care of children, beginning with one on prenatal care; that it has ready for the press the first installment of material which will appear as a statistical handbook, embodying the figures of the last United States Census relating to children, rearranged for convenient use by those interested in the welfare of children.

BABY-SAVING CAMPAIGNS

I wish to mention particuarly certain activities closely related to the inquiry into infant mortality. There will issue from the press within a few days the first of a series of annual bulletins on baby-saving campaigns, entitled "A Preliminary Report on What American Cities are Doing to Prevent Infant Mortality." While this material has of necessity been prepared hastily, it contains statements from various cities as to the precise methods of their work, including copies of baby-saving circulars in various foreign languages, so that it is really a handbook for health offcers desiring suggestions as to how to go to work in a given town.

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There is one important aspect of the efforts to reduce infant mortality which is brought out in this new pamphlet. It shows that, according to the census mortality figures for the registration area of the United States during the year 1910, nearly 10 per cent. of children dying under 1 year of age lived less than one day, and 23.5 per cent. lived less than one week. The same figures for the year 1911 show a still more distressing situation. In 1911, with an increased death registration area there was a decrease in deaths of children under one year; but those who lived less than one day increased not only relatively but absolutely—from not quite 10 per cent. in 1910, the figure has risen to 12.1 per cent. in 1911, while those living less than one week increased from 23.5 per cent. in 1910 to 27.4 per cent. in 1911. These figures show that the efforts to save babies through pure milk and more intelligent care have produced results in the general decrease in deaths of children, while throwing into marked relief the persistent increase in deaths during the first days of life, which these efforts alone cannot reach.

The New York Milk Committee in its 1912 report gives some pertinent figures in this connection. In that city the attempt was made to reduce, in one restricted area, this large percentage of infant mortality in the first days of life, by getting in touch with the expectant mother as early as possible during pregnancy. This campaign, begun in the summer of 1911, showed a reduction, up to the end of 1912, in this supervised area as compared with the borough of Manhattan, of 32 per cent. in deaths under one month and of 28 per cent. in stillbirths among 1,398 babies, with two deaths among 1,375 mothers.

Similar work for expectant mothers has been undertaken already in other cities, among which are Indianapolis, Providence, Baltimore, Chicago, St. Louis, Detroit, Richmond, Louisville, Milwaukee, Nashville, Cincinnati, Fall River (Mass.), Buffalo, and Boston.

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BETTER REGISTRATION NECESSARY

As stated above, the inquiry into infant mortality can be conducted only in communities where there is birth registration. Hence the bureau is of necessity cooperating in the campaign for better vital statistics and is especially bound to urge adequate birth registration because of the many ways in which accurate birth records may serve the ends of child welfare. Its first bulletin was on "Birth Registration, An Aid in Protecting the Lives and Rights of Children." The British Notification of Births Act provides for notification within thirty-six hours, and it has been said that no other one expedient has done so much toward lessening infant mortality in England.

I wish to speak of the cooperation of the bureau with health officers and other agencies. The city of Montclair, N. J., is undertaking an inquiry into its infant mortality and desired to use the same schedule which the government uses in its inquiries. We have therefore agreed with the health officer to furnish the schedules and to send an agent of the bureau to start the work.

It has been suggested above that the bureau is deeply interested in the question of the health of rural communities. We are therefore much gratified by the offer of the president of the University of Iowa to undertake, in cooperation with the bureau, the survey of a rural county in that state.

Of course, the ideal way to acquire knowledge of a community is not by a brief inquiry covering a stated period, but by a continuous and uniform system of civic accounting which shall take note of every child, from birth on, with such emphasis on special conditions as may be from time to time required. There are at present beginnings of inquiries of various types in various communities, from which we may expect some uniform method to develop. It is a hopeful feature that different communities are approaching this problem in different ways. The investigation undertaken by the Children's

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Bureau, which I have tried to outline above, is, of course, frankly experimental in several important particulars. We hope to see an inquiry in some city in which the city health department will follow the babies week by week through the first year.

So far as its resources permit, the bureau desires to place itself at the service of localities undertaking inquiries on the same lines which it is itself pursuing, and to cooperate in such a manner as to make the material of greatest possible advantage.

ABSTRACT OF DISCUSSION

Dr. C. F. Wahrer, Fort Madison, Iowa: A nation's greatest asset is its physical welfare, its health. Now, how can we bring these things before the people in a manner that will be acceptable and proper? Many people do not even know that it is a crime to allow children to stand up all night and tend machines when they should be in bed.

A short time ago six of us were appointed in our state to attend the World's Tuberculosis Convention at Chicago. As a test, I wrote to the governor asking him to give us fifty dollars apiece so that we could leave our work and attend this convention. He would not give us one cent. The following week the state gave thirty thousand dollars to a company of soldiers to dedicate a monument on a Southern battlefield. Everything for the dead, nothing for the living. One year we received only five thousand dollars for our state board of health, but that same year the state gave one hundred thousand dollars to put up a steel grandstand at the state fair grounds at Des Moines. They made this thing popular. We must make this Federal Children's Bureau popular. We must teach our politicians that child welfare is the most important subject for consideration when the budget is made up by the legislature.

Dr. H. D. Chapin, New York: I am sure that the medical profession will stand back of all this work and render all the help they can to this excellent bureau.

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ETIOLOGY OF ARTIFICIAL FEEDING

A PLEA FOR THE STUDY OF BREAST-MILK PROBLEMS

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The title of my paper expresses the desire that prompts its presentation—a wish to discuss, from actual clinical experience and from general observation, the reasons that lead up to a discontinuance of breast-feeding and which, therefore, bring about the institution of artificial feeding, and to prove that these reasons are usually fallacious. I shall not deal in statistics or cite classified groups of cases, but simply present the impressions which I have gained while following a clinical experience culled from private practice and from a comparatively large hospital clientele. The type of patients considered embraces a mixture of all classes, from the poverty-stricken alien who has but recently come to our shores to the infant of the rich surrounded with every luxury that the best city or suburban home can afford. The question is asked of every mother who presents an artificially reared babe or one on whom artificial rearing is being attempted, "Why did you take your baby from the breast?" In each instance—it matters not what the social status may be—the answer may be classified under one of three roughly made headings: (1) psychic, (2) educational, (3) economic or social. The divisions are not always distinctly separated, as a given reason may be embraced by one or more headings.

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PSYCHIC REASONS FOR DISCONTINUING BREAST-FEEDING

The psychic reasons may as a rule be referred to the maternal mental state at the time. How is this produced? The breast-feeding is usually discontinued through an innate fear either of not being able successfully to accomplish the act of nursing through not knowing how to go about it or through a fear that the product of the mammary gland is sufficient neither in quantity nor in quality to supply the nutritional demands of the infant. Either state is induced by a failure of the mother to recognize the possibilities of her own organism or through an instinct of unfounded anxiety for the welfare of her offspring. The reverse—a rare manifestation of innate selfishness—may be operative. In either of the two former instances the psychosis, if it may be so called, usually has no foundation in fact, or the temporary cessation of the lacteal flow is directly inhibited by the fear on which it depends. This inhibition of secretion finds its analogue in an identical effect on other secretions, notably that of the saliva, when the same etiologic factor, fear, is in evidence. Unfortunately, as will be pointed out later, the profession, by a tacit acquiescence in the probability of the actual disability or, in many instances, by actual encouragement in the idea, is often to blame, if not for the beginning of the trouble, at least for its continuance.

Here the physician loses a golden opportunity of becoming a legitimate psychotherapeutist and, by the same exhibition of indifferences or of actual carelessness in other psychasthenic states, permits the irresponsible exponents of questionable systems of mental therapy to succeed where he fails. It is my experience that this element of fear of not being able to accomplish the nursing function successfully is one of the most important factors in the discontinuance of breast-feeding, and I wish here to subscribe to the opinion that rarely, if ever, other things being equal, in an otherwise healthy women,

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can a permanent organic, irremovable obstacle to successful maternal nursing be demonstrated.

"I did not nurse my baby because I could not." "My milk poisoned my last baby and it died." Parenthetically, it may be stated that it was not the mother's milk that poisoned the baby, but in all probability the indifferent artificial feeding on which it was placed when the breast was unnecessarily withdrawn. "I never can nurse my babies because my milk dries up." These and similar replies are given to our queries as to why the breast-feeding was discontinued, and not one of them will stand the test of careful scrutiny or of future experience in a birth that is intelligently managed. Repeatedly in physicians' families have I seen instances in which I have been summoned, two or three months after unsuccessful artificial nourishment had been attempted to rear an infant because this element of fear had caused the breast-feeding to be discontinued; while in subsequent deliveries in which the care of the breasts had been placed in my hands and the mother had been properly fed and encouraged and morally supported and all external interfering sources had been excluded, a period of successful nursing has ensued which lasted nine or twelve months and could have continued longer had I so wished. Who among you has not had a similar experience?

The fear that the breast-milk is not the proper food for the infant often depends on the development of the signs of early indigestion—vomiting and green-curded stools, and a failure to gain immediately in weight. This will be referred to again, as it involves the physician's responsibility. Suffice it to say that green stools and vomiting do not constitute a bar to successful nursing, although I have found them to be among the most common causes that put to flight the physician in his half-hearted attempts to conserve the breast to the infant.

Among the better element a pretense of willingness to nurse is often made while at the same time every possible excuse is seized on by the mother in an effort to demon-

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strate her inability. The exigencies of social life, an inordinate love of pleasure, the confession of utter ignorance and helplessness, a desire to escape the obligations of maternity and to shift them on others, a cowardly exhibition of hysteria or assumed physical weakness underlie the plea for assistance. I know no class of patients more difficult to handle; I know no more disheartening task than to convince such mothers of their error. They must be handled diplomatically, yet withal sternly, the conscientious physician refusing to be ruled by them or to acknowledge the incorrectness of his position. One must appeal to their moral sense, the duty which they owe to their offspring, and point out to them the uncertainties and dangers of artificial feeding. The climax usually comes by the mother seeking advice from one of a more conciliatory mold. Few will yield when once they have determined to seek the services of the bottle until perhaps it is too late—in the presence of a fatal diarrhea, convulsions or pneumonia.

EDUCATIONAL REASONS

The reasons given that may be classified as coming under educational deficiency call into account both the physician's and the mother's responsibility. Infant-feeding, in spite of the so-called advances made in the science, is neither properly taught in medical schools, nor is it properly presented in most of the text-books, or practiced, as taught, by the leading dietitians in their hospitals or in private practice. I do not imply that the text-books fail to present the subject of artificial feeding thoroughly. On the contrary, the authors' attempts at comprehensiveness befog the issue. Too many views, too many theories and too many methods are still being taught. Students and practitioners when through perusing this or that authoritative work, or all of them, are left without the ability to apply practically what they have read. I was amazed recently when visiting the wards of one of the largest institutions of a certain city to find

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that the feeding cases were left by a distinguished chief in the hands of an inexperienced intern, who in his turn depended on a nurse of less experience. Stock mixtures, carelessly compounded, were fed. All the babies were doing badly. Answers to pertinent questions were evasive. Knowledge of metabolism, digestion and of the adaptability of the mixtures to the individuals was lacking. The wonder is, not that the average physician still refuses to be impressed with the value of scientific feeding, but that he ever attempts to follow out any case to its logical conclusion.

If this be true of artificial feeding, what may be said of the teaching and practice of breast-feeding? It is almost nil. The student and practitioner receive but little information as to how to interpret or to meet the signs of early indigestion, as to what are the best means of conserving the milk-supply and how to meet the problems of nutrition as they occur in the breast-fed. Authoritative opinion satisfies itself by stating that the best way to rear an infant is on the breast and, after demonstrating that the great yearly infant mortality depends largely on conditions attributable to artificial feeding, proceeds, without further comment on breast-feeding, to elaborate page after page of instruction on that means of feeding on which the greatest mortality depends. The paradox is real, since the conservation of human milk may be successfully practiced.

A breast-fed infant has colic: it is constipated or has diarrhea; it vomits; its stools are green and contain mucus and curds. None of these constitutes a bar to successful breast-feeding. With reference to vomiting itself I wish to state that, though it need rarely be so, it is one of the most common causes for withdrawing the breast. In all I have treated eight cases of pyloric obstruction, and in each instance maternal nursing had been discontinued because of the symptom—vomiting—the diagnosis of obstruction not having been considered A little care and use of the ordinary methods of physical

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diagnosis would have determined that the cause of the vomiting depended not on the mother's milk, but on an organic stomach affection.

On the other hand, vomiting dependent on faulty milk, as an excess of fat, or on overfeeding or too frequent feedings, does not constitute a valid contra-indication to the use of the breast. Dietetic, eliminative and mechanical treatment of the mother and the institution of common-sense hygienic measures will speedily correct the error.

An infant cries a great deal; it does not seem to get enough milk; it is said to be hungry after nursing; it does not gain in weight rapidly enough; the milk is too thin or too rich, scanty, or entirely absent. Only once in a healthy woman, in a case seen with Dr. Victor A. Loeb, have I met a real instance of an entire absence of milk.

"The mother is too nervous or too irritable." "She is too weak." "All her strength goes into the milk." These and many similar statements are made by the mother, or her friends, or the physician, or all, as an excuse to abandon the breast entirely or to institute partial artificial feeding. Just as vomiting and indigestion rarely constitute a valid reason, so too it may be demonstrated, though time and space forbid detailed consideration of each supposed contra-indication, that none of the many reasons stated are really legitimate bars to maternal feeding.

Abscess of the breast need not, in my experience, in every case indicate complete withdrawal of the breast. The size, location, duration and whether or not pus is secreted at the nipple are the determining factors. If, then, it can be proved that the statement with reference to these supposed contra-indications be true, where does the responsibility lie? What may be done to offset the pernicious tendency to credit these really trivial reasons with such great etiologic influence? Physicians must be educated first to recognize the possibilities of the breast,

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and they in turn must educate the mothers. The specialist must set the example for the practitioner. Instead of espousing this one's or that one's method of artificial feeding, let him see to it that he is known as an actual and practical advocate of breast-feeding. Let him refuse to place an infant on the bottle just because it is brought to him for this purpose, unless he is presented with a sound reason and one not founded on fear or fancy. Let him, by actual weighing experiments before and after each feeding, satisfy himself as to whether or not the infant is receiving sufficient food. He should consider the weight of the infant at birth, its present weight and its average gain before he determines the nourishment to be unfit. If it be found so, let him make an honest endeavor to rehabilitate the lacteal flow before confessing failure. The need for systematic propaganda is urgent, and to be given authoritative weight it should emanate from the Section on the Diseases of Children of the American Medical Association. We should cease arguing about percentages and calories, the etiologic importance of protein, fat and sugar, the rôle that bacteria play, nitrogen intake and ammonium output, the effect of calcium and potassium salts, the two-hour interval, or the four-hour interval, spasmophilia and the exudative diathesis. Let us cease teaching something to-day and decrying it to-morrow, but let us unitedly declare to the profession and to the motherhood of America that the only correct way to feed infants is on the breast, and then let us teach them how to do it.

The artificial feeding of infants has engaged some of the brightest minds of the profession. From the first attempts of Meigs and Pepper in America to adapt cow's milk to the digestive apparatus of the human infant down to the present day illustrious students, Jacobi, Rotch, Holt, Chapin, Finkelstein, Meyer, Keller, Czerny, Heubner, Rubner and others, besides a host of diligent laboratory workers, the trend of investigation has been along lines leading toward the solution of this problem.

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In spite of years of effort it has been but partially solved. Some uniform rules of feeding may have been enunciated, but no one food, or one method or one system of feeding has been determined as universally adaptable to the human infant. Have the years spent in patient toil and profound study been for naught? This query may be answered negatively if the profession realizes that it has not solved artificial feeding, and positively if it is not conscious of its failure and at the same time that it has not utilized the advantages of breast-milk feeding. Could one-tenth of the time and study devoted to the investigation of artificial feeding have been diverted toward the consideration of the conservation of human milk and of the causes which lead to its disuse and of the means of treating and meeting the gastro-intestinal and nutritional disorders of the breast-fed, infant mortality would have speedily and materially dwindled. The efficiency of breast-milk as a prophylactic against acute infectious diseases, rickets and scurvy, and its therapeutic value in many instances in combating a fatal issue, is well recognized but too seldom utilized. In private practice the incidence of an acute infectious disease is almost invariably the signal to discontinue breast-feeding. The mother is actuated by fear. The physician displays a lack of knowledge in conducting the feedings during the interim. Here is a case in point:

CASE 1.—An infant of 3 months with an acute empyema was admitted to the Philadelphia General Hospital. Bulging and redness appeared between the ribs. The empyema ruptured and the opening was enlarged and drainage instituted. After recovery, measles and, later, pneumonia of the opposite side developed, and still later the infant had convulsions. The treatment consisted alone of continuous breast-feeding and ordinary hygienic measures. No drug therapy was employed, and recovery was complete. To-day the baby is a fat, rosy specimen, with but the scar of the empyema remaining.

It would not have taken a great stretch of the imagination to prognosticate the fate of a poorly nourished, anemic, rachitic, indifferently artificially fed infant of

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this tender age under like circumstances. Breast-milk saved this baby's life and it will do likewise for others. We know well the effect of wet-nursing in many cases of decomposition(marasmus), which cannot be made to gain. The point is not that we do not possess the necessary knowledge—not that the case cited presents anything new—but that we fail, in our zeal to delve deeper into new methods of artificial feeding, to realize the treasure we possess in breast-milk, or, granted we do, we fail to put our knowledge to use. We are as

> one whose hand,
> Like the base Indian, threw a pearl away,
> Richer than all his tribe.

The indifference with which physicians sacrifice the human breast is nothing short of shocking, especially since they possess nothing quite so good to offer. Even to-day the woful lack of knowledge possessed by many practitioners as to how to adjust cow's milk to the needs of the individual is appalling. The cynical manner in which they regard the whole subject of scientific artificial feeding is disheartening. They are the chief offenders. They usually have many poor persons who seek their advice. It is among this class that the ravages of mortality are the greatest. It is these physicians we should reach. They are in need of propaganda which will educate them in the methods of breast-milk conservation.

According to my observations, maternal illness need not constitute a valid permanent contra-indication to breast-feeding in the majority of cases in which it is practiced. This has special reference to slight indispositions, colds, mild infections and minor operations. The ease with which the breast-milk may be rehabilitated can be witnessed daily, after a cessation of many days and even weeks. A wet-nurse was discharged from a case. The infant exhibited an idiosyncrasy to cow's milk. Persistent efforts were made for three weeks with the bottle. Rashes and gastro-intestinal symptoms

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developed (allergy). The wet-nurse was recalled. Her milk was found to be scanty and poor. Under forced feeding, general hygiene and the stimulus given to the mammary gland by the infant's lips, the fluid was reestablished to overflowing and reached a percentage of fat above 7 per cent., and measures had to be taken to reduce both quantity and quality. Eclampsia, nephritis, grave anemia, malignant disease, tuberculosis, certain forms of cardiac disease, pregnancy, epilepsy, insanity and prolonged infections are the type of illnesses which contra-indicate nursing.

The obstetrician, too, must assume his share of the responsibility in this war of conservation. He must be a breast-feeder enthusiast and should be capable, without the advice of the pediatrist, of handling the problem for at least the two or three weeks of his attendance. Beside his indifference to features other than those directly connected with the accouchement (care of the breasts, inquiry as to the condition of the nipples and the character of the milk, etc.), the greatest error into which he falls is to allow his patient to get out of bed too soon. This is usually followed by bleeding, which is deleterious to the quantity and quality of the milk. One obstetrician of my acquaintance is accustomed to give his patients a dose of Epsom salt daily after the third day. The result is that nearly all of his patients have bottle babies. This case illustrates what may be accomplished:

CASE 2.—One of his patients, the wife of a physician, had received a dose of Epsom salt daily for two weeks. Her milk was practically gone. She was anemic and undernourished, as was the infant at the age of 7 weeks. It was on the bottle in midsummer. The parents were much alarmed, as their first baby had died of intoxication (Finkelstein). Forced feeding, sensible hygiene and a tremendous amount of moral support rehabilitated the milk-flow and carried the infant through the summer and far into the winter with exceptionally satisfactory results.

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ECONOMIC REASONS AGAINST BREAST-FEEDING

Reasons for stopping breast-feeding, at once pathetic and difficult to overcome, are those which may be classified as economic or sociologic. No one denies that the poor and ignorant are in need of instruction in hygiene and general physical care. It may be pertinently asked, however, whether or not such instruction brings about desired results. Those who come into daily contact with disease the result of poverty, and endeavor to inject personality into their work and aspire to be more than medical men and to make their hospitals and institutions educational forces, often realize how hopelessly useless is the task. We are reminded of a saying accredited to Henry Ward Beecher that the most potent factor in the reformation of a hungry man is a sandwich. So these mothers, who are poorly housed and poorly fed, who must labor either within the home or outside of it, are best benefited by that advice which comes as actual material assistance to relieve, in part at least, their physical responsibilities.

On whom does the onus rest? On society itself and its representatives—the legislature. Free milk-stations, floating hospitals, clean milk and many other endeavors, providing what appear as luxuries to the poor, but which, as ordinary necessities, are secured, as if by divine right, or at least without the influence of visible external agencies, by those better situated, represent a well-intentioned but misdirected expenditure of thousands annually by the state and charitable organizations. Under our present social system, however, the necessity seems to exist. What else, other than charity, have we to offer to the mother who answers our demand that she nurse her babe by affirming that she must go to work; that she has not enough food to provide the ordinary fare, without considering the extra quart of milk which she must consume; that she must place her babe in a home or in a day nursery. Recently in Pennsylvania an act has been passed pensioning indigent mothers instead of

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separating them from their children by placing the latter in public institutions. While accomplishing some good its effect cannot be far-reaching, since it is practically a form of charity, itself a result of perverted social conditions and by its nature necessarily limited in its scope.

Further, it may be pertinently queried whether or not the permanent necessity for charity exists, and who is to determine who shall receive charity and who is to dispense it. In other words, why do poverty and its companion, ignorance, exist? Is it not possible that the error lies at the foundation of our social system, which denies to the worker his rightful share of that which his brawn has built, and gives the lion's share to the brain which conceives or directs? As a consequence his opportunities for education and self-uplift are limited. Does not charity simply represent a return by the individual or by the state of that which originally should have been given to the beneficiary as a product of his labor? If this be so, does it not appear patent that the necessity for charity not only does not exist, but that it is not humane and that, as before stated, it is makeshift and a blight on the state which encourages it?

One may ask, in what manner does this influence infant mortality in general and the problem of breast-feeding in particular? Its effect is potent largely for the reasons enumerated and because it hinders the independence of the nursing mother. It is recognized that however desirable an economic revolution which would benefit this class may be, its fruition and practical realization are not only problematic, but also far remote. The innate selfishness of human nature, the self-love, the exaggerated ego from which we all suffer, will indefinitely postpone the millenium of equal opportunity to partake of the natural resources of the earth and of the common well of knowledge. One fact stands out clearly, and that is that the nursing mother is performing for the state a duty not only as real but far more

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important than the artisan who is legally employed to build its bridges or to construct its highways. Why deny compensation to the one and not to the other? Let that mother who is devoting from nine months to one year of her time to the rearing of a future citizen of the commonwealth be relieved of material responsibility and be paid in actual cash for her services. This need not be regarded as chimerical but could, I believe, be put into practical operation and would redound to the good of the nation and would be a far more sensible expenditure of the public funds and certainly more humane than doled-out charity. Before legislators could be prevailed on to lend their influence to such a proposition the motherhood of the country and the profession are in urgent need of illuminating propaganda which would emphasize the advantages and possibilities of breast-nursing, the dangers and often the lack of the necessity for artificial feeding and the tremendous influence of the latter in maintaining the high-water mark of infant mortality. The burden of such a campaign of education is on that portion of the medical profession which is presumed to speak with authority on the matter of childen's diseases. I have appended the case histories briefly of a few of my personal experiences. They could be almost innumerably multiplied. Some appear commonplace and trite, but they teach a powerful lesson of the importance of breast-milk conservation. Each case lost to the breast adds its mite in influencing the sum total of the tremendous infant morbidity and infant mortality.

CASE 3.—Mrs. J., mother of four children, and healthy, refused to nurse because she said that her milk poisoned her last baby, causing its death. The present infant was immediately placed on artificial nourishment and finally passed through its first year after experiencing two severe attacks of spasms and one attack of severe digestive disturbance. This could have been avoided by breast-feeding.

CASE 4.—Mrs. M. had two children, both artificially fed. Both developed summer complaint and marasmus during the course of feeding, but finally recovered. The husband was a physician. The mother was told without reason that her milk

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was all water. She became pregnant again. From the day of conception, she was encouraged with reference to her ability to nurse her babe. After delivery intensity of moral support increased and she was well fed. Result: abundant milk-supply for ten months, and a healthy infant.

CASE 5.—Baby S., first baby, father a physician. Weight at birth 6½ pounds. Vomited every feeding. Bowels green and curded; anus excoriated. Mother very nervous. Diagnosis of probable pyloric obstruction made and advisability of weaning was being considered. Mother reassured. Pyloric obstruction eliminated. Milk contained high fat percentage. Mother dieted and purged. Told to ignore digestive symptoms. Small dose of strontium bromid and a little water administered before feeding. Baby continued to gain in spite of digestive symptoms, which gradually subsided. Present weight at six weeks, 10 pounds. Usual course in this case would have been to wean, with probable disaster on account of the approach of summer.

CASE 6.—Baby G., first infant. Mother well. Father a physician. Infant cried a great deal. Mother therefore convinced that she did not have enough milk and infant was starving. Very difficult to control. Finally convinced by actual weighing experiments before and after feeding and the progressive gain in weight.

CASE 7.—Baby L., father physician. Had lost one infant from summer-complaint on bottle. Mother during puerperium had received an ounce of Epsom salt a day and besides had inverted nipples and small breasts. Rose from childbed weak, anemic and with flabby breasts. Flow appeared about to be leaving. Regular nursing advised, forced feeding, with oatmeal and Southworth's soup.[1] Return of flow was gradual and the gain in weight tedious. I refused to wean on account of summer. Mother very nervous. Both parents, against their better judgment, frequently threatened to give artificial food. Flow finally established. Result: a fat, healthy babe.

CASE 8.—Mrs. W., mother of two children. Very stout. Breast well developed, but not so much glandular substance. Confined with present baby in hospital. Permitted to get up on ninth day and to leave hospital on tenth day. Result: severe uterine hemorrhage. Rest in bed and forced feeding only partially restored flow of milk. Finally had to wean in summer. Result: severe intestinal disturbances. Final recovery. Remarks: Fault lay with the accoucheur. All trouble could have been avoided.

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1. Southworth's soup consists of 2 tablespoonfuls of yellow corn-meal boiled one-half hour with 1 quart of water. Water of evaporation is made up by addition of hot water. Whole is salted and flavored to taste. About 1 quart a day is drunk.

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Thus, in a general way an attempt has been made to state the influences at work which have been responsible for the decadence of the habit of breast-feeding, and it would seem from my experience that the following conclusions are justifiable:

CONCLUSIONS

1. Breast-feeding is the only sane and safe method of infant-feeding.

2. The breast is too frequently sacrificed without cause.

3. Psychic influences are largely determinable so far as the mother is concerned.

4. Fear plays an important rôle in the etiology of artificial feeding.

5. The two most common examples of fear depend on the mother's belief in her inability to provide suitable nourishment and on the signs of early indigestion which are erroneously regarded as a contra-indication to the continuance of breast-feeding.

6. The profession of the country has not been sufficiently educated in the methods of dealing with nutritional and digestive disturbances of the breast-fed.

7. Medical curriculums are lacking in such instruction.

8. Too much time has been spent in studying the problems of artificial nourishment without very much progress or benefit.

9. Physicians resort too quickly to artificial feeding.

10. Economic and social reasons for discontinuing the breast are the most difficult to overcome and find their origin in the present social system.

11. Charity is but a makeshift to relieve the present state of error. It is unnecessary and not humane.

12. A mutation of economic and social conditions, while offering relief, is remote.

13. In the interim nursing mothers should receive actual cash compensation from the state.

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14. A campaign of education directed toward legislators, mothers and physicians is urgently needed and should be undertaken by the Section on Diseases of Children of the American Medical Association and the pediatric sections of allied organizations.

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ABSTRACT OF DISCUSSION

Dr. John Zahorsky, St. Louis: Most writers on the subject seem to agree that there are few mothers who are unable to nurse their babies. As an actual fact, I have great difficulty in private practice in keeping many babies on the breast, and I do not think that we can be as optimistic as Dr. Lowenburg that we can do this. I am excluding now any mother who has not enough to eat. I mean mothers of the better class who can pay for the best food and the best care. Many of them cannot nurse their babies in spite of all the psychotherapy that I can give and all the feeding methods that I can devise. The usual symptoms are acute indigestion and the baby does not thrive. The mother nurses the baby according to the ordinary rule. Nevertheless the baby does not thrive; it has severe dyspeptic symptoms that are not improved by lengthening the interval between feedings. What shall we do with these babies? They are very numerous. I have not been successful with these cases, and do not know anyone who has. I am collecting records of a large number of these cases which I hope some time to present. These babies are usually fed by supplemental or complemental feedings, and the mother soon finds that the baby does better, likes the artificial food better than the breast, and then she gives it more in spite of the doctor's advice. Why the mother's milk does not agree with the child we do not know.

When a mother asks my advice about putting her baby on the bottle, I say to her, "If you were going to have an operation performed on your baby in which there was a mortality of from 10 to 15 per cent. would you not consult several physicians before having to undergo the operation?" I then suggest that she consult two or three physicians before putting the baby on the bottle, since this has an equal mortality with an operation. We generally succeed in keeping these mothers from weaning the babies, but that does not always mean that the babies are fed.

Dr. C. F. Wahrer, Fort Madison, Iowa: I do not believe that a certain class of Dr. Zahorsky's patients come under Dr. Lowenburg's classification. I want to make a plea for those mothers who want to nurse their babies and can do it, but for some reason have been ignorantly advised. Within the last six months I confined two sisters about three and a

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half months apart. They both had a "Sary Gamp" for a nurse. She told the first sister that she could not nurse her baby because the milk was too thin. A few days after my last attendance the husband came to me and said the wife's breasts were hard and large. I said, "Have you been removing the milk?" He said, "Yes," I said, "What did you do with the milk?" He said, "Threw it away." I said, "What do you feed the baby on?" He said, "The bottle." Now in such a case the doctor must make his moral influence felt. I told him that they must feed this baby on its mother's milk or do without my services. If patients want to follow the advice of "Sary Gamp," who maintains that she has raised thirteen children and for that reason should give advice (although she has lost seven of them), they can do without my services. I told this nurse that she must not interfere any more or I would show her the door.

The same thing occurred when the second sister was confined. They also had "Sary Gamp." I told the mother that if she did not nurse the baby I would not come to the house again. Both these mothers are nursing their babies and are happy.

Dr. H. D. Chapin, New York: There seems to be some difference of opinion. There are some women who do wish to nurse their babies, but are physically unable to do so, although they are willing to follow all the directions that are given them.

Dr. H. W. Francis, Bancroft, Neb.: I live in a small town. I seldom have trouble in getting the mothers to nurse their babies. Occasionally we have a mother whose milk does not seem to agree with the baby. In my experience, the continued use of citrate of soda given to the baby at nursing time is often beneficial. I have continued this as long as three months without any harmful effect and it proves of benefit to the baby. It stops the colicky spells.

Dr. L. R. DeBuys, New Orleans: There is one thing that Dr. Lowenburg may have forgotten to mention which I think should be emphasized, namely, the care of the breast and the care of the nipples before confinement. It is absolutely essenital. I recently saw a mother who was extremely anxious to nurse her baby. The baby was 11 days old. It was torture for her to try to nurse him, her nipples were so fissured. In a few days under the proper management the nipples were healed and she resumed nursing her baby without any pain or inconvenience. It is essential to have good nipples in order to have mental rest and effectual encouragement.

We meet two classes of mothers, those who want to nurse their babies and cannot, and those who do not want to and can. In the case of the latter I think that we are perfectly right in taking the position that Dr. Wahrer has taken. In

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those cases in which the mother wants to nurse her baby and cannot, my practice has been, if she is supplying a little milk which is not causing any digestive disturbance, to weigh the baby before and after nursing to determine what the baby is getting and make complemental feedings to meet the requirements. I have frequently found that the mother can nurse the baby for six or seven months in this way.

In an instance in which the mother was giving 4 ounces in twenty-four hours, by this method, in the course of three months she was giving 20 ounces of milk; the baby, of course, was having complemental feedings. Incidentally, the mothers in these cases were in good circumstances.

There is a tendency to wean the baby if the stools are not perfect, that is, are not normal custard stools. We must remember, in this connection, that it takes some weeks, two or three, sometimes a month, for the baby to become adapted to the breast and the breast to the baby.

I have had a rather unique experience in connection with an abscess of the breast. The breast had to be operated on. I allowed the baby to continue to nurse the healthy breast; and during the period when the diseased breast was being treated surgically I used puppies to nurse it, and in this way kept the breast functionating. After the breast had entirely healed and there were no organisms in the milk, the baby was put back to that breast, which continued to functionate satisfactorily.

Dr. Collins H. Johnston, Grand Rapids, Mich.: I have not found many mothers who do not wish to nurse their babies. Many mothers have not been taught that the mortality of bottle-fed babies is eight or ten times that of breast-fed babies. In my lectures to nurses I always emphasize the fact that there are but two contra-indications on the mother's part to nursing a baby. One is advanced heart disease, the other pulmonary tuberculosis. There are, of course, other occasional conditions in which we may, if necessary, advise weaning, but these two classifications will cover the great majority of cases.

I think also that physicians are frequently too careless in permitting mothers to wean their babies. We do not always sufficiently realize the difference, not only in the first year of life, but in later years, between breast-fed and bottle-fed babies. We all know that the death-rate among bottle-fed babies in the first year of life is much greater than among breast-fed babies, but we do not all realize the great influence breast-feeding has on the growth and development of children in the first five or ten years of life.

I saw some interesting statistics pertaining to this at the International Hygiene Exhibition in Dresden two years ago. In twenty-four families with one hundred and nine

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children, all breast-fed, at the end of at least five years of married life, not one of the children had died, while every one of the thirty-three families in which babies were all bottle-fed had lost one or more children during the same period.

Especially interesting were the results in families in which some of the children were breast-fed and some bottle-fed. In seventy-nine families of this class investigated there was a group of twenty-nine families with eighty-five breast-fed and one hundred and nine bottle-fed babies. At the end of eleven years all of the breast-fed children were alive and 57 per cent. of the bottle-fed babies were dead. In another group of thirteen families with forty-eight breast-fed and twenty-three bottle-fed babies, at the end of ten years all of the breast-fed babies were alive and all of the bottle-fed babies were dead.

We must not lose sight of these results of breast-feeding in infancy. The two main periods in which mothers wean their babies before the natural time for doing so are in the first month and in the fourth or fifth. The causes of weaning in the first month are vomiting, diarrhea, etc.

I often tell mothers that I do not care how many stools the baby has in the first month of life, whether one or two or twenty a day, nor how much the baby cries, provided it gains in weight. It frequently takes several weeks for a baby's stomach to adjust itself to mother's milk and during this time more or less gastro-intestinal trouble is bound to result. If the mother concludes that this is due to some imperfection in her milk and weans her baby on this account, a great mistake is made. I believe that the occasions when a baby should be weaned on account of some fault in the mother's milk are rare. When, after a thorough trial, it is found that a baby will not thrive on breast-milk, the fault is usually with the baby and not with the mother.

Dr. Frank C. Neff, Kansas City, Mo.: I think that Dr. Lowenburg's criticism of the medical profession is well founded in many ways. It does not apply, however, to men who are practicing pediatrics.

A custom which I have followed, and which I understand from Dr. Smith is followed in Boston, is to secure the breast-milk whenever it can be obtained from healthy women. Often the mother is willing to nurse the baby, but she thinks that she has not enough milk and does not know what else to do but give it the bottle. In this case, it is my practice to give supplemental feedings: to find out how much the baby is getting by weighing it before and after nursing and then to get supplemental milk from relatives or friends or any healthy mother who has more milk than she needs. When this milk is not procurable in any other way I have

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resorted to advertising in the daily paper, offering to pay a healthy mother so much a week for so many ounces of milk, she to come to the house as often as necessary and have the milk drawn out to be kept on ice and used subsequent to each nursing—not to take the place of any nursing. The baby should be nursed at the breast as much as possible and then fed this breast-milk from the bottle. I should like to ask you to try this method. It is surprising what a small amount of breast-milk some babies thrive on. This will solve many problems in cases in which the milk is insufficient for complete feeding.

Dr. E. O. Benson, Chicago: In the great majority of cases, there is no difficulty in inducing the mother to nurse the baby. In fact, the great majority of mothers among the better classes are anxious to nurse their babies. They have heard that there is a large proportion of mothers who cannot nurse their babies, and they are afraid that they may be in that class.

Years ago, when percentage feeding was first introduced, it was thought that a panacea had been found, but experience has taught us that there is no substitute for mother's milk, and this knowledge has come to the reading laity. As Dr. Gengenbach mentions, the nursing possibilities of women are influenced by their mental emotions and by artificial surroundings. A large proportion of women among the poorer classes take it for granted that they should nurse their babies. They have nothing else to think of but the care of their houses, their husbands and babies. Some mothers have difficulty because they are underfed, others because they are overfed. We must avoid too rich food as well as too poor food. In certain rural communities in the old country, in which mothers eat sour milk, old bread and little butter, possibly meat once a week, and otherwise subsist on cereals and gruels, you will find a large proportion of healthy babies and a large proportion of mothers who are able to nurse their babies.

I do not believe that physicians are such great sinners as Dr. Lowenburg seems to intimate, but we must put this matter strongly to physicians in order that they may advise every mother to nurse her baby.

Some babies, even after the best efforts, must be put on artificial feeding. Dr. Lowenburg rather condemned the time spent in studying artificial feeding, but many babies come to us after they have been weaned without the advice of a physician. When we leave a case, after confinement for example, we tell the mother to come to us when the baby is in difficulty. She does not do it. She first takes the advice of the neighbors or the grandmother, and then, after the child's digestion has been damaged by experiment, she comes

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to the physician and he has to meet the problem of artificial feeding. The number of these cases is large and the physician is not responsible for them.

Dr. H. Lowenburg, Philadelphia: My experience does not permit me to agree with Dr. Zahorsky as to the inability of many mothers to nurse their babies, nor with the importance he places on the occurrence of dyspepsia in breast-fed babies. Possibly I meet just as many cases of indigestion and green stools in the breast-fed as he does, but I have come to regard them as of no importance and pay absolutely no attention to them. I meet green stools, curds and colicky babies, but, if the child continues to gain in weight—you will find that most of them do—I think that we should pay no attention to the character of the stools at all. I believe that from the cradle up infants require a little wholesome neglect. I think that they should not be fussed over too much. I think that we should pay less attention to the importance of dyspepsia in the breast-fed and think that all women with mammary glands can nurse. The patients that come under our observation who will not be able to nurse their babies will then become fewer in number. I do not say that women are not willing to nurse, but I do say that many of them are actuated by fear that they cannot, or that their milk does not agree with the child. It is important, as Dr. Zahorsky suggests, to make a systematic study of the stools of the breast-fed, to learn, if possible, just why it is that these infants do have green movements, because I believe that almost every one of them does.

It is important that the breasts should receive attention during the whole puerperium. Every other physical defect of the mother should be noted and corrected as well.

Dr. Wahrer mentioned, as an etiologic factor in the discontinuance of breast-feeding, the interference of relatives. This is often carried to extremes, especially by those who have had little if any experience, as when a maiden aunt who never gave birth to a child advises against nursing. Trained nurses are often responsible for unasked-for advice which leads to a discontinuance of the breast-food. I should not tolerate any interference from the nurse in a matter of this kind. Feeding and the care of the breast is in the hands of the physician, and the nurse should know it from the beginning. I criticize both the pediatrist and the general practitioner. So many of these cases occur among the poor and ignorant. Their physicians actually profess to know nothing about breast-feeding and still less about bottle-feeding, and yet the first thing they do when they meet colic and green stools is to put the baby on some patent food. I think that this criticism of the general practitioner is just. I think that the reason he is at fault, and this is where the

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pediatrist deserves censure, is that the pediatrist sets him a bad example by being a little too much in a hurry to put the child on the bottle. I will not put a baby on the bottle without a good sound reason.

Dr. Chapin's proposition is a fair one. I usually get at the women psychologically as best I can. I start out with the proposition that they can nurse and explain to them the advantages which will accrue to their infant, themselves and their husbands. Then I convince them of the importance of weighing the child before and after nursing to demonstrate to them that they have enough milk. Weekly weighings are insisted on, not oftener, to show them that their babies are gaining. Sometimes I will admit that I cheat a little bit in the beginning and tell the mother the infant has gained even if it has not. I think it is best to do this in order to encourage her. Soon the gain is inaugurated in nearly every case. Then I study the milk. If I find any ingredient at fault I increase or diminish it by regulating diet, fluids, laxatives and exercise. I always flood them with water in the beginning whether the milk is good or bad. This increases the total quantity and aids the infant's digestion. The quality can be controlled later. I wish again to emphasize the importance of the psychic element. There is only one type of woman that occasionally appears to be practically hopeless, and that is the very fat woman who has more fat than glandular substance.

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LEUKOCYTE COUNTS IN PNEUMONIA AND CEREBRO-SPINAL MENINGITIS

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INTRODUCTORY

It has long been recognized that the leukocytes perform an important function in the struggle against bacterial invasion of the organism, and many important contributions have been made to this subject. But very few of these studies have been devoted to infants and young children.

The pneumonias of infants have been particularly slighted, and that fact led to the undertaking of this work, more especially as our clinical experience has caused us to ponder as to the correctness of the assertion that lobar pneumonia was infrequent until after the second year. It is, however, true that most of the cases that come to necropsy under the second year are of the mixed type; these, however, are rarely primary or uncomplicated cases.

To make this work of practical interest, cases of lobar and bronchopneumonia as well as complicated cases have been selected, fifty in all. It was hoped that some information of value for clinical use might be gained when the blood is examined in its relation to the clinical picture in the individual case; first, as to the virulence of the invading strain; second, the reaction on the part of the organism; third, the presence of a true or pseudo crisis, and fourth, the onset of complications.

Interest in the study of the blood in epidemic cerebrospinal meningitis was stimulated by a chance reading of Rusca's article, published some two years ago, on differential blood-counts in a series of nine cases with six deaths and three recoveries, treated without the use of serum. All of his cases were adults, with the exception of one child, aged 5. It was hoped that some information might be gained whereby these children might be saved repeated spinal puncture for diagnostic purposes

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and unnecessary injections of serum, through the light gained by some of our untoward experiences immediately following such procedures.

Conclusions, so far as they are possible, are based on the absolute counts of the different cells, rather than percentages, the latter being unreliable and misleading in their interpretation, due to the rapid changes in the total counts as influenced by different clinical phases and by treatment.

A discussion of averages in the various groups of cases has also been avoided because such information is also misleading. Reliable information can only be gained by a careful study of the blood-findings with reference to the clinical manifestations in the individual case.

BIOLOGY OF THE LEUKOCYTES

There are two general theories regarding the development of the leukocytes, dividing hematologists into two distinct schools. One, under the leadership of Ehrlich, considers that the polymorphonuclear neutrophils, eosinophils and basophils are derived only from their corresponding marrow-cells, and that there is never a transition from the lymphocytes to the polymorphonuclear forms. Lymphocytes, according to this school, develop from the various lymphoid organs, some even from lymphoid tissue in the marrow, and pass through the various stages of lymphoblasts, large lymphocytes and small lymphocytes, but never into the types of cells with granular cell bodies.

The large mononuclears and transitional cells, according to this school, are separate entities derived from the marrow, and the transitional cells probably are derived from large mononuclears and in turn are transformed into neutrophils. Pappenheim, Naegeli, Weidenreich and the majority of observers now accept this theory.

The other theory, which is held by the Russian school under the leadership of Uskow, and which is also supported by Grawitz, holds that the neutrophil cells are frequently derived from the lymphocytes, and that the so-called transitionals often represent a stage between these two types of cells. Practically all authorities agree that true lymphocytes do not act as phagocytes, although it has been demonstrated that they do in some way hinder the development of the bacterial infection in the body.

In the development of the neutrophils, Arneth's contention is that their progress from the myelocyte is marked by changes in the nucleus,

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it being first indented and bent and finally lobulated; the mononuclear forms, therefore, representing the younger and the multinuclear forms the older type of cells. The granules also are more numerous in the older cells. In the younger cells, the granules are apt to be first basophilic and later oxyphilic.

Arneth's contention, supported by numerous clinical observations, is that in various diseases the differential count of the neutrophils, according to his scheme, very often shows an abnormal predominance of the younger form of cells, which he calls a shifting to the left in his blood-picture. That this shifting is very characteristic and represents a definite reaction on the part of the organism to the infection and that a study of these changes forms a valuable basis for the differentiation of diseases and knowledge of their clinical course and subsequent history.

In my work I have accepted the classification of Ehrlich, first, because of its simplicity, and second because, with our present limited knowledge of the life history of the white cells, it is the more generally accepted theory of to-day. I have, however, digressed to this extent, in that I am showing the eosinophils in separate curves so that the return of the eosinophils during the period of convalescence of the patients may be more graphically represented. This has very little bearing on the total counts because of their limited numbers, and, as will be shown, the rarity with which there is an absolute increase in these cells.

It is likely that the eosinophils are derived from the large mononuclear leukocytes, but just how, we do not know. That there is a distinct relation between the eosinophils and digestion, and that a distinct and established relation exists between infections and the eosinophils there can be little doubt. The eosinophils are in the vanguard of the leukocytes, they disappear very early in infectious diseases, and their return is late.

EHRLICH'S CLASSIFICATION OF LEUKOCYTES

Lymphocytes, large and small.

Size: Small, 5 to 8 microns. Large, 8 to 10 microns.

Number: In adult, 22 to 25 per cent. of leukocytes, 1,500 to 1,700 per cubic millimeter. In infants, 40 to 60 per cent.

Nuclei: Usually central, stain deeply, one or two nucleoli present.

Protoplasm: Usually basophilic, but in old cells sometimes acidophilic.

The larger cells possess an irregular staining nucleus, with a chromatin network and a faintly granular margin of protoplasm. There may be exceedingly large cells in the blood of infants which when stained with the polychrome methylene blue-eosin stains show a distinct granulation in about 50 per cent. of their number.

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Large Mononuclear Leukocytes.

Size: Two to three times the size of red-blood cells.

Number: In adult, 1 per cent. of leukocytes, 70 per cubic millimeter.

Nuclei: Large, oval, eccentrically placed, staining much less intensely with the basic dyes than the lymphocytes.

Protoplasm: Weakly basophilic, without granules (Ehrlich stain). Nodal thickenings are present and they show a granulation with the Giemsa and triacid stains.

Transitional Cells.

Size: Resemble large mononuclear leukocytes, but are often larger, being the largest in the blood.

Number: In adults, 1 to 3 per cent. of leukocytes, 70 to 210 per cubic millimeter.

Protoplasm: Stains quite deeply with Ehrlich's stain and often presents a very few neutrophil granules.

Polymorphonuclear Neutrophils.

Size: 10 microns.

Number: In adults, 70 to 72 per cent., 4,900 to 5,040 per cubic millimeter. In infants, 18 to 42 per cent.

Nuclei: Stain deeply.

Protoplasm: Takes a faint acid stain.

Granules: Neutrophilic. Many may be on the nucleus. When migrated these are the ordinary pus cells.

Eosinophils.

Size: The same size or a little larger than the polymorphonuclears.

Number: In adults, 2 to 4 per cent, 140 to 280 per cubic millimeter.

Nuclei: As a rule more faintly staining.

Protoplasm: Slightly more abundant.

Granules: Eosinophilic, but do not lie upon the nucleus.

Mastzellen.

Number: 0.5 per cent., 35 per cubic millimeter.

Nuclei: Polymorphous and faintly staining.

Protoplasm: Acidophilic.

Granules: Basophilic and are not affected by the triple stains.

In the normal differential count there is considerable variation among authorities. This is largely due, however, to the fact that there is no unanimity of opinion as to what types of cells are included in the various groups.

NUMBER OF LEUKOCYTES IN BLOOD-PICTURE IN INFANTS AND CHILDREN

Da Costa gives the following figures for the total and differential leukocyte counts in infancy:

Small lymphocytes, 50 to 75 per cent. First year, 10,000 to 12,000.
Large lymphocytes, 6,000 to 14,000. Sixth year and upward, 7,500.
Polymorphonuclear neutrophils, 28 to 40 per cent.
Eosinophils, 5 to 10 per cent.

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Morse's figures for leukocyte counts in infants under 2 years are as follows:

Total, 10,000 to 14,000.
Polymorphonuclears, 35 to 45 per cent.
Small mononuclears, 40 to 50 per cent.
Large mononuclears, 10 per cent.
Eosinophils, 1 to 5 per cent.

TABLE 1.—LEUKOCYTE COUNTS IN 150 HEALTHY INFANTS AND CHILDREN AT DIFFERENT AGES. (D. RABINOWITSCH)

Age, Years	Total Leukocyte Count	Polymorphonuclear Count		Large and Small Lymphocytes		Transitionals		Eosinophils		Mastzellen		Large Mononuclear Neutrophils
		Total	Per Cent.	Total	Per Cent.	Total	Per Cent.	Total	Per Cent.	Total	Per Cent.	
1 to 2.	6,500	1,950	30	3,965	61	162	2.5	234	3.6	25	0.4	
2 to 3.	7,350	2.570	35	4,080	55.5	183	2.5	426	5.8	28	0.4	
3 to 4.	6,400	2,270	35.5	3,360	52.5	192	3	397	6.2	20	0.3	Varies from 1.0 to 3.3 per cent. of total.
4 to 5.	6,560	2,500	38	3,410	52	138	2.1	426	6.5	26	0.4	
5 to 6.	7,100	3,585	50.5	3,020	42.5	192	2.7	213	3	28	0.4	
6 to 7.	6,800	3,370	49.5	2,650	39	184	2.7	367	5.4	34	0.5	
7 to 8.	6,325	3,005	47.5	2,275	36	278	4.4	405	6.4	32	0.5	
8 to 9.	6,140	3,070	50	2,240	36.5	172	2.8	344	5.6	32	0.5	70 to 200
9 to 10.	6,160	3,170	51.5	2,220	36	154	2.5	333	5.4	21	0.3	
10 to 11.	5,850	3,220	55	2,020	34.5	146	2.5	257	4.4	23	0.4	
11 to 12.	6,790	3,600	53	2,340	34.5	163	2.4	510	7.5	34	0.5	
12 to 13.	6,250	3,530	56.5	1,970	31.5	194	3.1	300	4.8	37	0.6	
13 to 14.	6,890	3.995	58	2,135	31	248	3.6	296	4.3	28	0.4	
14 to 15.	7,000	4,585	65.5	1,750	25	217	3.1	266	3.8	19	0.3	
15 to 16.	8,000	5,440	68	1,840	23	256	3.2	328	4.1	50	0.6	

Rabinowitsch (Table 1), from his studies, came to the following conclusions:

1. Healthy children between the ages of 1 and 15 years average between 6,000 and 7,000 leukocytes, approximately the same as adults.

2. Polymorphonuclear neutrophils increase gradually from 30 per cent. in the first year to about 70 per cent. in the fifteenth year.

3. Lymphocytes decrease from 60 per cent. in the first year to about 30 per cent. in the fifteenth year.

4. The reversal of the percentages of neutrophils and lymphocytes occurs usually about the sixth year.

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5. Eosinophils average between 4 to 6 per cent., but vary greatly in different children at the same ages.

6. Transitional cells average approximately 2 to 3 per cent., not varying greatly at the different ages.

7. Mastzellen, about 0.3 to 0.6 per cent. Frequently absent.

8. Large mononuclear neutrophils, 1 to 3.3 per cent. About the same at the different ages.

TABLE 2.—LEUKOCYTE PERCENTAGES IN CHILDREN (KARNITSKY)

Age, Years	Total Leukocyte Count	Percentage of Neutrophils	Percentage of Lymphocytes	Percentage of Eosinophils
1 to 2.....	12,500			
2 to 3.....	9,400	40.7 to 30.2	60 to 75.6	Varies Greatly. 3.8 to 8.6
3 to 4.....		40.3 to 32.1	58 to 45	
4 to 5.....		39.7 to 34	53 to 45	
5 to 6.....		59 to 35	52 to 32	
6 to 8.....	7,900	51 to 37.3	52 to 35	
8 to 10.....		62.6 to 40	46.6 to 24	
10 to 12.....		66.6 to 43.7	39 to 22	
12 to 14.....		63.5 to 47.6	40 to 23	

TABLE 3.—LEUKOCYTE PERCENTAGES IN CHILDREN (CARSTANJEN)

Age.	Neutrophils, Per Cent.	Lymphocytes, Per Cent.	Eosinophils, Per Cent.	Transitionals, Per Cent.	Large Mononuclears, Per Cent.
1 to 6 mos.	34.5	50	3.5	10.5	0.5
6 to 12 mos.	40.8	49.2	0.7	8.2	0.9
1 to 2 yrs..	41.9	47	3	7.5	0.4
2 to 3 yrs..	48.2	38.4	3.9	8.7	0.7
3 to 4 yrs..	52.6	33.2	5.7	7.9	0.5
4 to 5 yrs..	61	25.8	6.3	7.2	0.3

The predominance of lymphocytes in the hematology of the infant is an exceedingly interesting and important question. The probability is that the lymphocytes do predominate in the normal infantile blood, but it should be demonstrated that these children are perfectly normal at the time of examination. It is certain that in the acute infections the lymphocytes are early replaced by the polymorphonuclear lymphocytes.

It has been with considerable reluctance that I have convinced myself of the continued high lymphocyte count as described in the various

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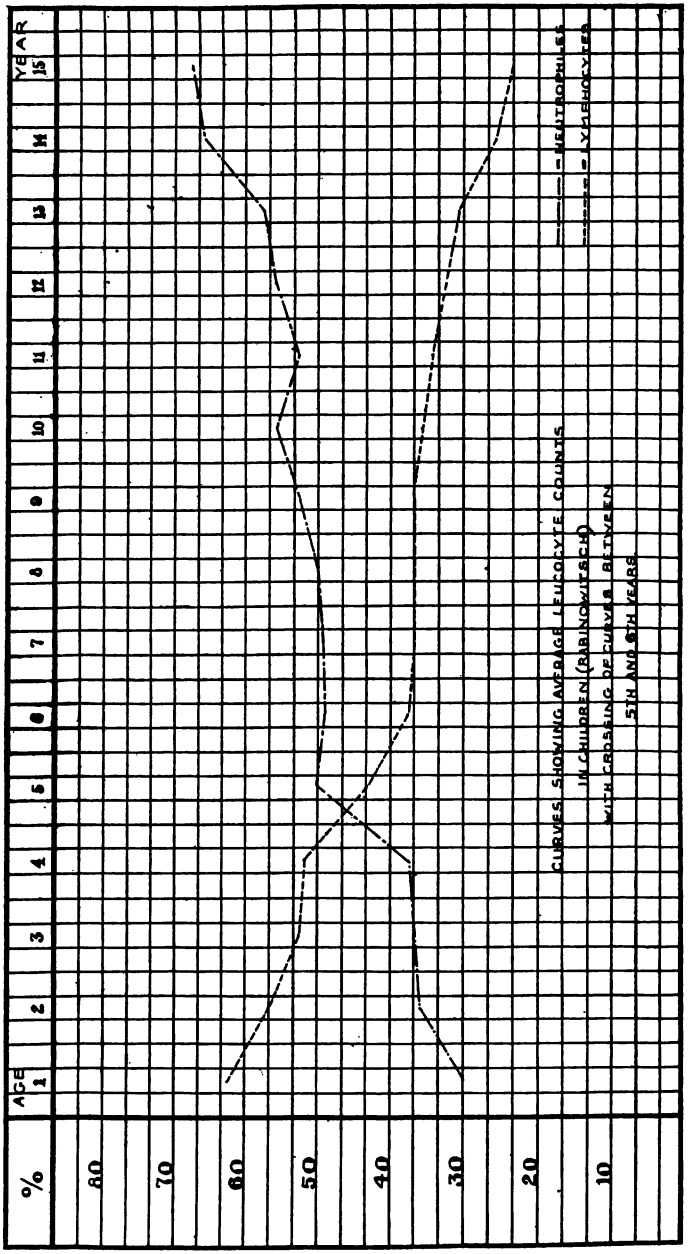

Chart I.

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monographs on the blood in late infancy and early childhood, in the face of the work here presented. Certain it is that early in normal childhood the curves simulate the adult counts; but nevertheless it is quite true that many illnesses more common to early than to adult life do involve the lymphatic system, and thereby stimulate these organs to cell functions which dominate the blood-picture. I desire to present seemingly convincing evidence of the similarity of the reaction to acute infection in infancy, childhood and adult life.

Leukocytosis in children is of extremely common occurrence, often arising from causes of the most trivial character and developing to a greater degree and much more rapidly than in the adult. It is, therefore, to be regarded with less significance than when it occurs in the mature. Usually the polymorphonuclear cells are chiefly involved in the increase, but the inclination of the blood of children to revert to the embryonic type appears to cause in many cases a disproportionate increase of lymphocytes in relation to the other forms, this peculiarity being especially true of the various pathological leukocytoses not due to the acute infections. Physiological leukocytosis in children usually affects chiefly the polymorphonuclear neutrophil cells.

BLOOD EXAMINATIONS IN EPIDEMIC CEREBROSPINAL MENINGITIS

Rusca, in his article, gives the results he obtained from repeated blood examinations in cerebrospinal meningitis and quotes from them in nine cases; six of which were fatal and three in which there was recovery, and all of which were treated symptomatically without the use of serum. This group of cases is of especial interest when comparisons are made with my group of cases—all of which were treated with Flexner serum—both as to the effect of the serum and of anaphylaxis on the blood-picture in the cases which ended in recovery and in the fatal case. In this article Rusca refers to work on the blood in epidemic cerebrospinal meningitis by Türk, Schindler, Zand and others. They all found a more or less marked leukocytosis with a predominance of the polymorphonuclear leukocytes, but they are not agreed as to the prognostic importance of the disappearance of the eosinophils, some believing that the disappearance was of ill omen, others believing it to be a good sign. The therapy practiced in Rusca's cases consisted of repeated lumbar punctures, large doses of sodium salicylate, ice packs, caffein and nutrient enemas. He finds in all cases a more or less marked leukocytosis.

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The polymorphonuclear leukocytes show the greatest increase. The lymphocytes are increased in some cases, decreased in others. With each exacerbation there is an increase in the percentage as well as the absolute number of neutrophilic leukocytes and the reverse is true in improvement. With the lymphocytes the opposite prevails. This results in a separation of the two curves during the recrudescence and an approximation and often a crossing during convalescence. In Rusca's three cases which ended in recovery, the curves crossed. In the first case, this occurred on the thirty-sixth day of the illness. In the second, it followed very rapidly on the first lumbar puncture, which was made during the fourth week of illness. In the latter case, before lumbar puncture, the polymorphonuclear leukocytes were 12,741, the lymphocytes 2,132, with no eosinophils or basophils. Two days later there were 3,227 polymorphonuclears, 3,500 lymphocytes, 35 eosinophils and 28 mast cells. He states that had serum been used in this case, it would have been erroneously credited with this marked change. He also notes a measles-like rash in the first days of the illness in this case, whose occurrence had been previously described by Curtis and who states that it is of good import. Block, Curtis and others suggest that the presence of a hemorrhagic rash, in contradistinction to the measles-like rash, is of bad import. In Rusca's third case, crossing of the curves occurred on the ninth day. In all of these cases the crossing of the curves was due to two factors: an absolute decrease in the polymorphonuclears and an absolute increase in the lymphocytes. The eosinophils were markedly increased in two of his cases during convalescence, in each to 6.5 per cent. In the third case they never were more than 216 per cubic centimeter, or 2.5 per cent. In the fatal cases the eosinophils were either very much decreased or absent during the acute stage and were always absent before death. The mast-cells vary, being absent in some of the cases during convalescence, increased during the course of one of his fatal cases, but always absent before death.

He concluded that at the beginning there is always a leukocytosis associated with an absolute increase in the polymorphonuclear leukocytes. During the course of the illness a separation of the polymorphonuclear and leukocyte curves occurs and during convalescence an approximation. In all of his cases that ended in recovery, the curves crossed, and were associated with a normal or increased eosinophil count. In the lethal

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cases there was no crossing of the curves and the eosinophils and mastzellen always disappeared before death.

Of the following six cases of my series, five recovered and one died:

CASE 1.—Carl D., entered the hospital Oct. 21, 1912, and was discharged Nov. 16, 1912, after recovery. Although the child was well on the morning of the 18th, toward evening he began to complain of sudden stiffness of the neck which steadily grew worse. On the morning of the 19th he suffered with a severe headache which persisted up to the time of his entering the hospital. Epistaxis occurred on the 20th, and vomiting and delirium on the morning of the 21st. Temperature, not taken with any regularity, had been present from the onset.

Examination and Course.—On entering the hospital the child was restless, talking loudly and crying incessantly in delirium. The head was held rigid and retracted, the cervical region was tender and a slight opisthotonus was present. The pupils were dilated and regular, the right slightly larger than the left, and reacted sluggishly to light. Photophobia was marked. The heart action was irregular and tumultuous. The extremities were somewhat rigid. Brudzinski and Kernig signs were marked. The knee-jerks, tendo Achillis and planter reflexes were all markedly exaggerated. Ankle clonus was present. The triceps and wrist-tap reflex was brisk. Babinski, Chaddock, Gordon and Oppenheim tests were all positive on both sides. Tache cérébrale was present. The abdominal and cremasteric reflexes were brisk.

October 21. Spinal puncture was performed immediately on entrance and about 30 c.c. of turbid fluid was removed under distinctly increased pressure. Twenty-five c.c. of antimeningitis serum were injected. An examination of the spinal fluid showed all globulin tests positive. Direct cell-count showed 40,000 per cubic millimeter. The differential count gave polymorphonuclears 72 per cent., small lymphocytes 24 per cent., large lymphocytes 4 per cent. Gram-negative intra- and extracellular diplococci were present, the proportion being about the same. Forty-eight-hour cultures showed meningococci. The blood-culture was negative.

Oct. 22. The child was more rational. Headache still persisted and photophobia was marked. The temperature ranged from 100.6 to 102 F. During the evening the pulse became very small and irregular, respirations shallow and irregular. Spinal puncture was repeated and a still turbid fluid under increased pressure was obtained. Thirty c.c. of antimeningitis serum was injected. The white count was 24,800.

Oct. 23. The condition was markedly improved. A slight headache and stiffness of neck were still present. Twenty-five c.c. of turbid spinal fluid under increased pressure was removed and 30 c.c. of serum injected. The temperature ranged from 100 to 103.8 F. The white count was 25,000.

October 24: The general condition was still improved, though headache and photophobia still remained. No spinal puncture was made on this day. The pulse was more regular. The white count was 14,000.

October 25: A slight opisthotonus and photophobia were still present. The maximum temperature was 99.8. The white count was 10,000. No spinal puncture was made.

October 26: The general condition was improved. The maximum temperature was 99.2.

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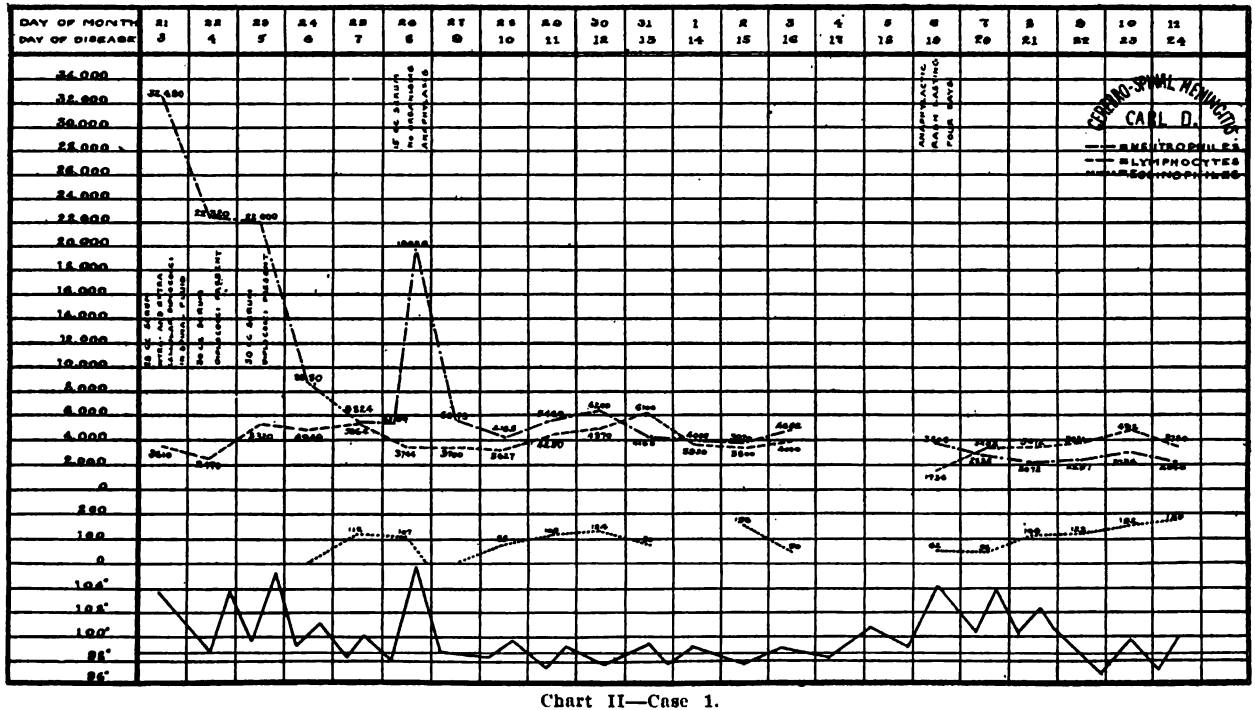

Chart II—Case 1.

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October 27: Twenty-five c.c. of almost clear spinal fluid was removed and 15 c.c. of serum were injected. The temperature was 98.6 in the morning, 103.6 in the afternoon. The pulse was 104. The patient complained of severe pain in the head and neck. The white count immediately after puncture was 10,700 and seven hours later 23,400.

October 29: Physical examination showed the head still held rigid and retracted and some tenderness present in the cervical region. The lower extremities were flexed on the abdomen and the reflexes remained the same as on admittance.

November 6: The afternoon temperature was 103.6, pulse 126, respirations 36. No signs of meningeal irritation were present.

November 7: Edema developed during the day about the eyelids and a few circumscribed erythematous lesions and wheals were found scattered over both legs.

November 8: The temperature was 100 to 102.2. There was no change in the general condition.

November 9: The maximum temperature was 100.

November 14: The patient was up and about, feeling fine. The reflexes were normal. No Kernig, Brudzinski nor Babinski signs were present.

November 16: The patient was discharged.

Further laboratory reports on the spinal fluid were made as follows:

October 21: Intracellular diplococci before serum injection, 8; after injection, 9. Extracellular diplococci before injection, 12; after injection, 11.

October 22: A differential count of the spinal fluid showed polymorphonuclears, 80 per cent.; small lymphocytes, 20 per cent.

October 23: Direct cell-count of the spinal fluid showed 29,000; the differential count showed polymorphonuclears, 82 per cent.; small lymphocytes, 15 per cent.; large lymphocytes, 3 per cent.; meningococci numerous; intercellular organisms, 32; extracellular, 28. Cultures were negative at the end of forty-eight hours.

October 27: The spinal fluid was clear; no organisms were found.

Case 2.—Edna D., sister of Carl D., entered the hospital Oct. 21, 1912, and was discharged Nov. 16, 1912, after recovery. On the morning of the day of admission the patient complained of severe headache, which was accompanied by pain and stiffness of the neck and increasing stupor. She had been feverish and vomited several times.

Examination and Course.—The child was somewhat stuporous, with a retracted head and slightly rigid neck; the pupils were small, equal, with normal reaction and no ptosis or strabismus; the knee-jerks were markedly exaggerated, especially on the right side; the plantar and Achilles tendon reflexes were active, the Babinski sign was marked on the right side, suggestive on the left; there was no ankle clonus. Gordon, Chaddock and Oppenheim reflexes were positive on both sides; Kernig and Brudzinski signs were pronounced.

October 21: The temperature on the day of admission was 101.8 F.; pulse, 136. Twenty-five c.c. of turbid fluid under pressure were withdrawn by spinal puncture and 15 c.c. of serum were given. The white count was 28,800.

October 22: The child was rational, but complained of headache. Another lumbar puncture was made and 15 c.c. of serum were given. The highest temperature was 100.6, the pulse 108 to 120. The white count was 26,000.

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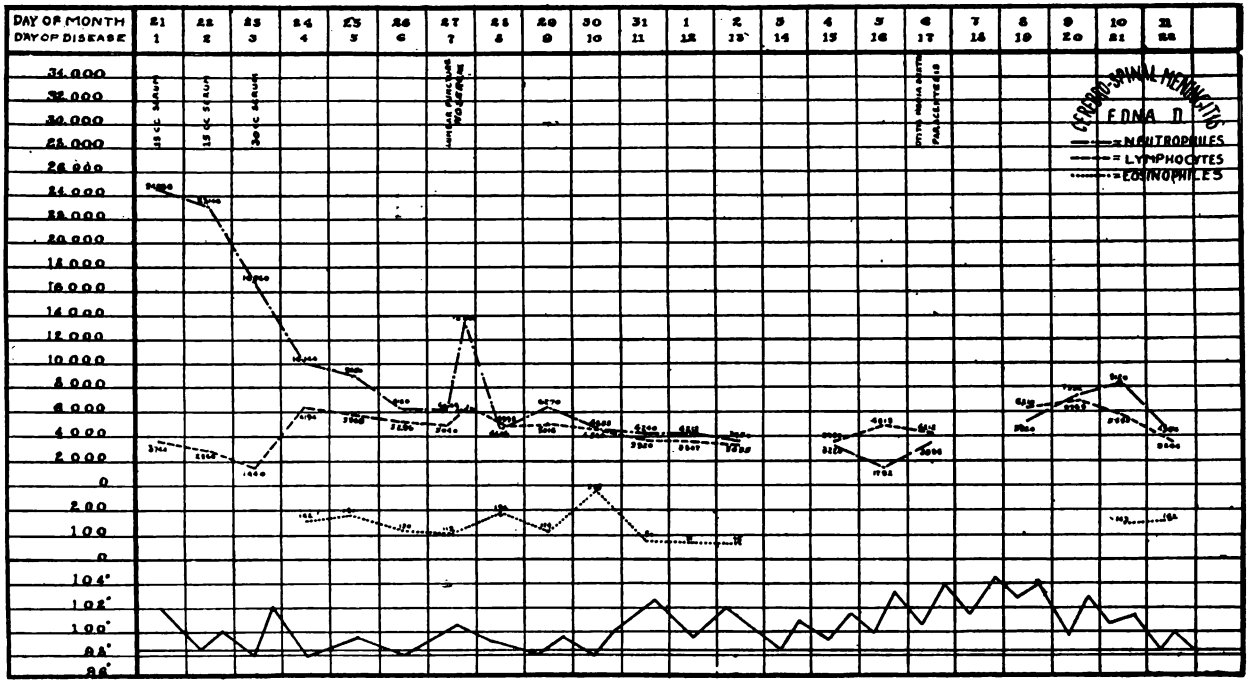

Chart III—Case 2.

October 23: The maximum temperature was 102, pulse 132. Twenty-five c.c. of turbid fluid were removed under increased pressure and 30 c.c. of serum were injected. The white count was 18,000.

October 24: The patient was much improved, though the head was still retracted. The temperature was 99, pulse 112, the white count 16,200.

October 25 and 26: The improvement continued, though a slight headache still persisted. The temperature was about normal.

October 27: The lumbar puncture was repeated. The fluid was clear, but pressure was still increased. No serum was given. The maximum temperature was 100.4.

November 5: The temperature rose to 103. The heart and lungs were negative.

November 6: The temperature ranged from 102.6 to 104.8. The right tympanic membrane was found injected and bulging and a paracentesis was done. The white count was 7,600.

November 7: The temperature ranged from 103.2 to 104.6. No signs of meningeal irritation were present.

November 11: The maximum temperature was 101 and the condition of the patient was good.

November 16: The patient was discharged with temperature and reflexes normal.

The laboratory report of the spinal fluid was as follows:

October 21: Globulin tests were all positive. Direct cell-count of spinal fluid 60,000, with 80 per cent. polymorphonuclears, 16 per cent. small lymphocytes, 4 per cent. large lymphocytes. Before serum injection 21 intracellular and 19 extracellular Gram-negative diplococci were present.

October 22: After injection, 23 intracellular and 17 extracellular diplococci were present.

October 23: The direct cell-count of spinal fluid was 28,000, with 77 per cent. of polymorphonuclears and 23 per cent. small lymphocytes. Intracellular meningococci, 28; extracellular, 29.

October 27: The spinal fluid was clear. No organisms were present.

Case 3.—Edward W., aged 7, was taken ill on March 10, 1912. Sixteen hours after the onset, 30 c.c. of serum were injected. Twelve hours later the white count of the spinal fluid was 48,600. The child was delirious at that time. The temperature was 106. March 11, another 15 c.c. of serum were given and the same quantity March 13. The white count on that day was 16.520.

March 15: The child was worse. The temperature reached 105. Seventy-five c.c. of spinal fluid, containing intracellular and extracellular diplococci, were removed. The white count was 23.050.

March 16: The maximum temperature was 102, possibly due to serum disease. Thirty c.c. of serum were given.

March 17: Thirty c.c. of serum were injected. The temperature reached 102 again to-day. The white count was 16,520.

March 19: The patient's condition was much improved. The temperature was normal. The white count was 14,400. At the end of two and one-half weeks slight anaphylaxis showed without rise of temperature. A myositis developed also, with flexure of the right thigh on the abdomen, which lasted five weeks. The patient recovered.

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CASE 4.—Leonard B., aged 4, was taken sick on March 17, when vomiting occurred for the first time. March 18, the patient vomited and had a convulsion; was drowsy, but rational and had no pain. The urine on that day showed 3 per cent. of sugar.

Examination and Course.—A slight internal strabismus showed. Brudzinski and Kernig signs were positive. Babinski, Gordon and Oppenheim reflexes were negative. The reflexes were slightly exaggerated. The pupils were equal and reacted normally.

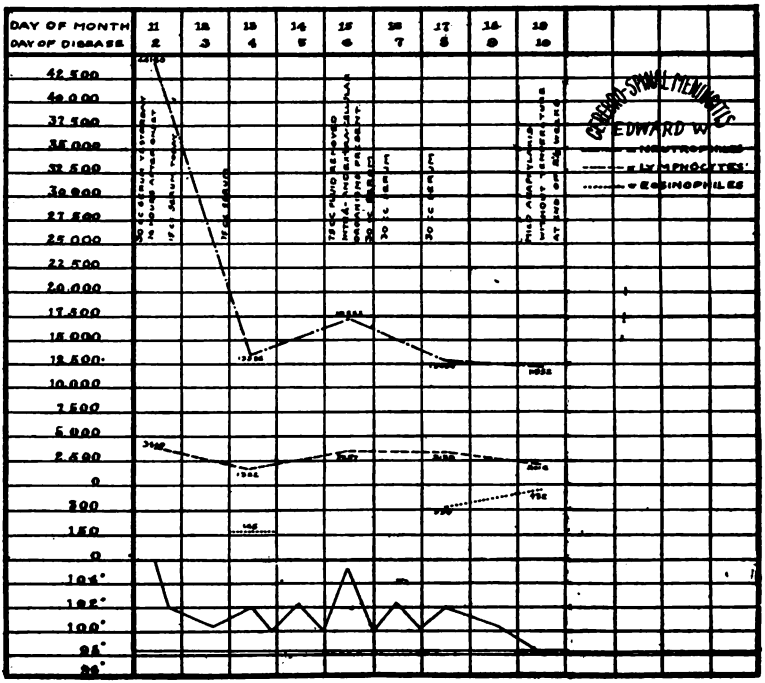

Chart IV—Case 3.

March 19: Thirty-five c.c. of very cloudy spinal fluid were withdrawn. Thirty c.c of serum were given, which was followed by complete respiratory paralysis, with weak heart and no pulse. Artificial respiration and peripheral stimulation restored the child, after which repeated convulsions occurred. Extracellular diplococci were present in the spinal fluid. A differential count showed 33 cells present, of which 6 per cent. were small lymphocytes and 94 per cent. polymorphonuclears. The white count was 31,650.

March 20: Thirty c.c. of serum were given. The white count was 25,600.

March 21: Fifteen c.c. of serum were given. The patient was in constant convulsions. The white count was 26,200.

March 23: The patient was slightly better. The white count was 27,800.

March 24: Decided improvement was manifest. The patient was now semi-conscious. The white count was 16,900.

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March 26: The condition was worse. There was spasmodic twitching of the lower extremities. Another 15 c.c. of serum were given. The white count was 19,400.

March 27: The child was comatose. The white count was 22,720.

March 28: Condition was again much improved. The white count was 16,400.

March 29: The patient was semiconscious to-day. The white count was 17,800.

March 30: The white count was 10,000. The patient recovered.

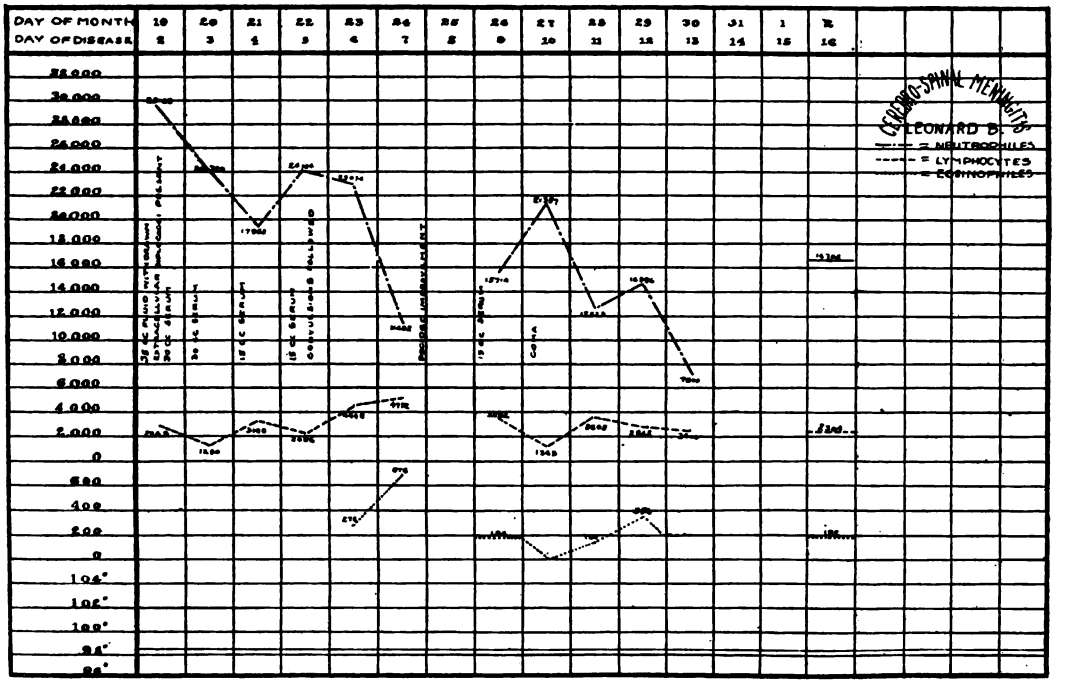

Chart V—Case 4.

CASE 5.—Edward T., aged 2 years and 9 months, was admitted May 11, 1912, and discharged May 30, 1912, after recovery. The onset was two weeks antecedent to the admission, and was with vomiting, fever, drowsiness and one convulsion. Constant retraction of the head had been present.

Examination and Course.—The patient was a robust child with marked retraction of head and constant convulsive twitching of the upper extremities. The temperature was 105, pulse 144. A true rotary nystagmus was present and there was a slight purulent discharge from the nose. The reflexes were increased. Babinski, Chaddock, Gordon, Oppenheim and Brudzinski signs were all present.

May 11: Thirty c.c. of spinal fluid were withdrawn and 30 c.c. of serum were given. The globulin test was positive. Seventy-four polymorphonuclears showed per cubic centimeter. Gram-negative intracellular diplococci were present. A twenty-four hour culture showed no growth. The temperature gradually declined to normal on the fourth day in the hospital, then suddenly rose to 105. This was

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accompanied by pain in and then a discharge from the left ear. Five days afterward the temperature again reached normal. A day or two later the child developed measles.

The white count on admission was 22,800, the next day 11,400, the fourth day 7,200 and the sixth 9,900.

CASE 6.—Harriet S., aged 23 months, was admitted May 15, 1913, and died May 20, 1913. The child was taken ill two weeks before admission with convulsions, which lasted an hour, stiffness of limbs, abdominal pains and vomiting, and since had had constant fever.

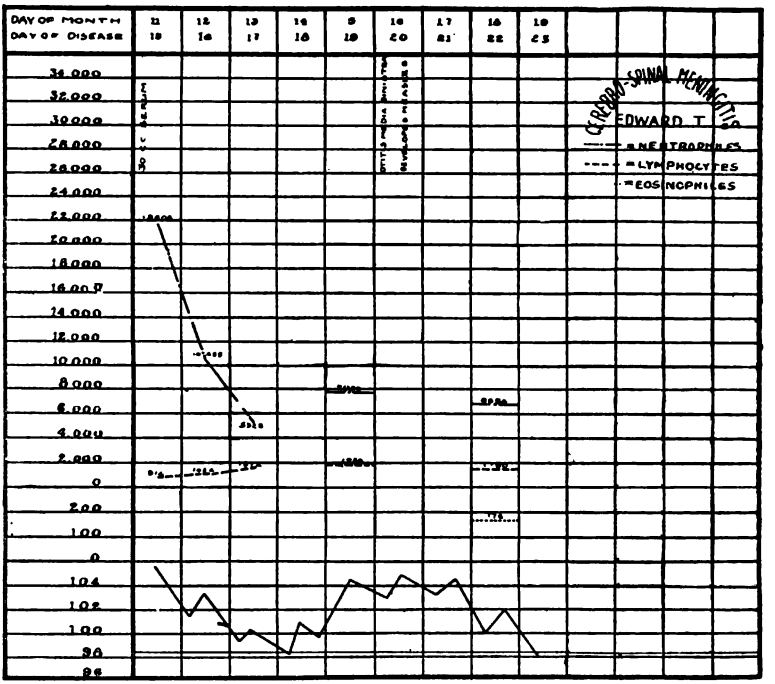

Chart VI—Case 5.

Examination.—The patient was a well-nourished child with a retracted head, slight opisthotonos and almost constant twitching of the extremities. The eyes showed a double ptosis and a fine rotary nystagmus, but no strabismus. The spine was rigid and slightly opisthotonic. The reflexes were exaggerated. Kernig, Chaddock, Gordon, Babinski and Oppenheim signs were present.

May 15: Twenty c.c. of cloudy, yellowish spinal fluid were removed. This contained intracellular diplococci. Globulin tests were positive; 1,000 cells per cubic centimeter, with 95 per cent. polymorphonuclears and 5 per cent. small lymphocytes. Fifteen c.c. of serum were injected. Ten c.c. of bloody spinal fluid were removed during the evening and 15 c.c. of serum were given.

May 16: Four c.c. of spinal fluid were removed and 15 c.c. of serum were given.

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May 18: A few small bright red spots were present on the neck, chest and abdomen. Fifteen c.c. of serum were given.

May 19: The condition was grave. There was Cheyne-Stokes respiration.

May 20: Large and small irregular blotches and wheals were present on the face, body, limbs and mucous membranes. A dry lumbar puncture was made. Fifteen c.c. of serum were given. The temperature ranged during the illness from 100 to 103.4, reaching 106 just before death. The white count rose from 33,800 on the day of admission to 40,200 three days later, then dropped gradually to 30,300 just before death.

SUMMARY OF FINDINGS IN PRESENT SERIES OF CASES OF EPIDEMIC MENINGITIS

1. Total Leukocyte Counts

In Case 1, the patient was seen on the third day, suffering from a very virulent form of meningitis associated with delirium, extreme rigidity, etc. The total count before treatment was 36,100.

In Case 2, the patient, aged 5, a sister of the patient in Case 1, who was seen during the first eighteen hours of the illness, was still rational, and had a total count of 28,800.

In Case 3, the patient, aged 7, was seen within twelve hours of the onset, and had a total white count of 48,000. The child was at this time wildly delirious. In this case the count was not taken until twelve hours after the first 30 c.c. of serum were given.

Case 4, in which the symptoms were very violent, was seen twenty hours after the first vomiting, and four hours after the first slight convulsion. This patient remained in coma for fully three weeks. The total white count was 31,650 within twenty hours of the first attack of vomiting.

Case 5, in which the patient was aged 2 years and 9 months, was apparently a subacute case with a history of vomiting two weeks previously and a gradually increasing opisthotonos, rigidity, etc., and entered the hospital with white count of 22,800.

Case 6, in which the patient was aged 2, was very severe, the patient entering the hospital with a history of convulsions two weeks previously, with which were associated opisthotonos, rigidity, diarrheas, etc. The white count was 33,800.

As will be seen, all of these cases presented a high leukocyte count. In the first four, all of which recovered and which were seen very early in the attack, the number passed 30,000 and went as high as 48,000. In

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the fifth case, a subacute one, resulting in recovery, the count was 22,800, and the last case, also subacute, but with fatal issue and a complication of diarrhea, showed 33,800. Therefore, we may conclude that all of our series were associated with a marked leukocytosis.

2. Percentage of Neutrophils

Of this high leukocyte count, the polymorphonuclear neutrophils average in the first case, 90 per cent.; in the second, 80 per cent.; in the fourth, 92 per cent.; in the fifth (subacute case), 82 per cent., and in the sixth (subacute case with death), 84 per cent. This conclusively shows that the polymorphonuclear neutrophils were the predominating cells in these first counts made before serum was given.

3. Course of Neutrophils

In Case 1 the polymorphonuclear cells, numbering 32,490 before the first serum, dropped to 22,300 within twenty-four hours after the first 25 c.c. were given and to 8,680 after 85 c.c. and within three days after the first treatment. In this case, the injection was guided by the finding of both intra- and extracellular organisms up to the time of the injection of the third dose of serum.

Case 2. The 24,568 neutrophils present on the first examination dropped to 23,000 after the first 15 c.c. of serum, to 16,500 after the second 15 c.c. and to 10,500 after the injection of a total of 60 c.c.

Case 4 showed a polymorphonuclear count of 29,000 before the first 30 c.c. and 24,300 following the first. On the fifth day the count again rose to 24,000 and was associated with marked convulsions. The seventh day there was a decided improvement, with a fall to 11,492, two days after the patient had had his last serum, a total of 90 c.c. Another 15 c.c. was given on the ninth day, which was followed by more marked spasm and more intense coma, with a rise of the neutrophils to 15,714. Following this, there was gradual improvement and they again dropped to 7,600.

Case 3, with 44,160 polymorphonuclear leukocytes before the serum was given, dropped to 13,386 within forty-eight hours after the injection of 60 c.c. However, on the sixth day there was again a rise to 19,822, and on examination of the spinal fluid, it was found to contain both intra- and extracellular organisms. Sixty c.c. were again injected, 30

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c.c. on each of the two subsequent days and the neutrophil count fell to 11,952. This was followed by complete recovery. On the sixth day with the return of the organisms, there was a rise in temperature to 105.5, followed by lesser rises on the two succeeding days to 102, but associated with steadily decreasing polymorphonuclear and lymphocyte counts and the reappearance of eosinophils following their disappearance with a recurrence of the organisms on the sixth day.

Case 5, complicated by the occurrence of measles, entered the hospital with a count of 19,608 neutrophils. Thirty c.c. of serum were given and within forty-eight hours the count dropped to 5,322. On the fifth day in the hospital the neutrophils reached 7,920, and on the sixth day the child developed measles. The count made on the eighth day and when the measles eruption was at its height, showed 6,864 neutrophils, and for the first time a return of the eosinophils. This case is of importance because of the influence which the measles may have had on the count. Rusca and others have described a measles-like eruption in some favorable cases, but they do not state specifically whether or not these cases were true measles. In passing, it is of interest to note that in this severe subacute case, in which the child was undoubtedly exposed to measles after the onset of the meningitis, the influence of the measles on the course of the latter was favorable, if it influenced it at all. It might also be noted that with the onset of the prodromata and subsequently the rash of the measles, the temperature rose to 104.5, and reached this point on three subsequent days. As in the cases with an anaphylactic reaction, in which the temperature rose to a considerable degree, there was no reaction on the part of the white count.

Case 6 showed 29,744 neutrophils on entering, steadily rising to 35,778 on the third day in the hospital, notwithstanding the fact that 60 c.c. of serum had been given. At this point the child was in deep coma. Following this, another 30 c.c. of serum were administered, and the neutrophil count dropped to 26,576. Death ensued without the overcoming of the high neutrophil count. This case might be likened to some of the pneumonia cases which show an extremely high count in the neutrophils in the presence of very virulent infection and fatal result.

Case 1 had the highest total lymphocyte count, 6,100 on the tenth day following the serum, as compared with 3,610 on the day the serum was given.

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In Case 2, the highest lymphocyte count was 6,400 on the seventh day after the serum, as compared with 3,744 before the serum was given. In this case they rose as high as 6,768 on the twenty-first day. In these two cases with the disappearance of the organisms, the total lymphocytes became about double the number seen before serum was administered. While the percentage of large lymphocytes in Case 1 was reduced to 1 per cent. before reaction to the serum, in the lighter case, Case 2, they did not decrease below 4 per cent. In Cases 3 and 4, although there was an absolute increase in the lymphocytes, slightly more marked in Case 4 than in Case 3, in neither of these cases was there any great increase, the highest point reached in Case 4, being 4,732, and in Case 3, 3,840.

Case 5, the one complicated with measles, showed a decrease from the normal rather than an increase at any time.

Case 6. In this, as with the neutrophils, there also was a constant increase in the absolute lymphocyte count, not due, however, to any marked increase in the percentage of the cells as seen in the blood, but to the marked general increase in the white count. Therefore, in all of these cases, except the one complicated by measles, we see a true lymphocytosis, however, in no case a marked one.

5. Relation of the Neutrophils to the Lymphocytes During Convalescence

In Case 1 we see a very close approximation of the two curves on the fifth day following serum and the crossing of the curves on the tenth day.

In Case 2 we again see a very close approximation on the fifth day following the serum and the crossing of the curves on the eighth day.

In Cases 3, 4 and 5, while the curves are not carried along for a sufficient time to allow of a very close approximation of the curves, there was a tendency on their part to converge as soon as the result of the serum was manifest. This was not so in Case 6, in which there was a tendency to diverge up to the time of death.

6. Eosinophils

Only once in all of my counts did the eosinophils reach 4 per cent. of the total cells counted, and only occasionally as high as 3 per cent. In all cases during the acute state and before the serum reaction, there was a complete disappearance of eosinophils. In Case 1, they did not

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reappear until 90 c.c. of serum had been given, and they disappeared again for two days following the reaction on the fifth day. Following this they remained constant, the count ranging from 50 to 180 to the cubic millimeter, but at no time during the twenty-one days of this case did they reach what might be considered a normal count.

In Case 2, they also remained absent until the fourth day and until 60 c.c. of serum had been given. Following this, they remained constant, ranging from 1 per cent. to 3 per cent. during the following nine days, when they again disappeared for several days during the development of an otitis media. The highest count was 3 per cent, or 285 cells, on the tenth day following the first serum. More commonly they ranged between 80 and 196, never passing what might be considered the normal range in the healthy child.

In Case 1, although they did not entirely disappear during the period of anaphylaxis, the lowest count during the whole convalescence was found at this time.

In Case 4 they remained absent until 90 c.c. of serum had been given, on the fifth day they reached 278, and, with a decided improvement on the sixth day, they numbered 676. As in Case 1, with the injection of serum on the eighth day there was a marked increase in the leukocytes with a corresponding decrease in the lymphocytes and a disappearance of the eosinophils. They again reappeared during the period of improvment and remained quite constant throughout the convalescence.

In Case 3 they appeared on the third day following the serum, again to disappear with the recurrence of the meningococci in the spinal fluid, remaining away until the eighth day, when they reappeared, ranging in number from 330 to 532 and staying constant during the balance of the time.

In Case 5, the one associated with measles, the eosinophils remained absent until the eighth day after the serum, when they appeared, 176 to the cubic millimeter.

In Case 6, with a fatal issue, there never was any reappearance of the eosinophils during the stay in the hospital.

CONCLUSIONS AS TO EPIDEMIC MENINGITIS

1. All of my cases were associated with a marked leukocytosis.

2. The neutrophils were always the predominating cell before the administration of serum.

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3. There was a moderate but absolute lymphocytosis in all cases, most marked during convalescence, except in the one infected with measles. It was most marked in the fatal case.

4. There was always an approximation and in some a crossing of the neutrophil and lymphocyte curves during convalescence.

5. Eosinophils disappear absolutely very early in the infection and do not recur until convalescence has set in. With recrudescence and the reappearance of organisms in the spinal fluid, they again disappear. Following the withdrawal of spinal fluid and the injection of serum, there may be an increase in the total white and neutrophil count with a decrease of eosinophils, but in our experience, although the late anaphylactic reactions may be associated with a rise in temperature, the polymorphonuclear cells remained low and eosinophils present. Although there may be a distinct increase occasionally, at no time in our series of cases was there a marked eosinophilia. Their presence and reappearance in our cases was always associated with signs of good omen.

6. Every case must be studied individually with reference to the findings presented, not only as to the presence of meningitis, but the possibility of secondary infections.

7. Cellular blood examinations have no diagnostic value. Lumbar puncture is the source of accurate information.

8. Much can be learned as to prognosis by regular examination of the leukocytes. A drop in the neutrophil count with an increase in the lymphocytes and a reappearance of the eosinophils indicates a good prognosis. An approximation of the neutrophil and lymphocyte curves is of good import.

9. The leukocyte count is of most importance as an indicator for further prognostic punctures or the need of serum. An increase of the neutrophils and temperature with disappearance of the eosinophils should always lead to further spinal puncture.

10. The temperature and cerebral reactions often associated with late anaphylaxis can be interpreted by careful leukocyte counts, and the further injection of serum, with its accompanying dangers, may be avoided. The neutrophils remain low and the eosinophils usually are present during this stage.

11. Serum should always be injected by the gravity method.

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Believing that a careful study of the general blood-picture, as well as the differential cell counts, would be of considerable value in the differential diagnosis of pneumonia, in forecasting the prognosis as to recovery and the development of complications, and also in establishing the diagnosis of the type of pneumonia in young infants, that is, the differentiation between lobar and bronchopneumonias, a series of fifty cases of pneumonia of different types and associated with various complications were selected for this work. The cases have been arranged in a series of tables, according to type and complications, and each one is accompanied by an individual summary of the salient features of that group. Several interesting conclusions of practical value in the classification, differential diagnosis, prognosis and complications of the pneumonias have been reached by these analyses.

LOBAR PNEUMONIA

Series 1.—Uncomplicated Pneumonia Under 1 Year of Age

SUMMARY OF TABLE 4

The two cases in Table 4 were the only ones of uncomplicated pneumonia in my series, and both were of the lobar type, which is worthy of notation, because of the frequent classification of pneumonia under 2 years as bronchopneumonia. In Case 1, the neutrophils never fell below 62 per cent., nor the combined lymphocytes above 35 per cent. As in the older infants and children, the eosinophils returned with convalescence. In Case 1, there was tendency on the part of the lymphocytes to remain high after beginning convalescence. In the second case the neutrophils were 82 per cent. at the height of the infection.

These findings are of interest because of the marked reaction of the polymorphonuclear leukocytes to the pneumococcus infection, just as one would expect in older healthy children, notwithstanding the normal lymphocytosis of infant blood. These findings differ from Cases 32, 34 and 35, aged, respectively, 5½, 6 and 2 months, which were secondary pneumonias in rachitic and anemic infants. In all three of these cases there was a distinct lymphocytosis. In Case 37, a ten-week-old infant with a double lobar pneumonia and a secondary empyema, there was again the distinct reaction seen in the infections of older individuals, namely, 53,800 leukocytes, with 76 per cent. of neutrophils, 24 per cent. of combined lymphocytes and no eosinophils.

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This series of six cases leads me to the following conclusions:

1. Primary pneumonias of early infancy are commonly of the lobar type.

2. Young infants react to infections with a high neutrophil count, the same as is seen in older children.

3. Eosinophils remain absent in young infants during the height of infections.

4. Bronchopneumonias are usually either secondary pneumonias or occur in infants with low vitality.

SUMMARY OF SERIES 2, TABLE 5

1. The primary pneumonias of Series 2 were of the lobar type.

2. In well-nourished infants between the first and second years, the larger the amount of lung tissue involved, the greater the tendency to a high leukocytosis.

3. Anemic and poorly-nourished infants usually show lower total leukocyte counts.

4. The drop in the total white counts as well as the neutrophil count following convalescence is usually less rapid than in older children. This is interesting when we consider the normal high lymphocyte count of infants.

5. In uncomplicated cases the eosinophils usually returned on the day of the crisis or in one to four days following, but rarely in numbers exceeding the normal limits. In the complicated cases, they were slow in reappearing.

CONCLUSIONS AS TO SERIES 3 AND 4, TABLE 6

1. All were lobar pneumonias.

2. A true leukocytosis was present in all cases, highest in the uncomplicated cases.

3. Neutrophils were always the predominating cells before the crisis, and in all uncomplicated cases fell rapidly after the crisis.

4. In all cases there was a true lymphocytosis comprising both large and small cells, the small cells, however, predominating. Following the crisis they rapidly dropped to near the normal in most instances.

5. In the nineteen cases of these two series the eosinophils returned in Case 13 (with two previous attacks of pneumonia) four days before

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TABLE 7.—SERIES 5. BRONCHOPNEUMONIA

Days Since Onset	Date of Blood Examination	Temperature, F.	Total White Count	Differential W					
				Polymorphonuclears	Per Cent.	Small Lymphocytes	Per Cent.	Large Lymphocytes	Per Cent
5	2/23/13	103	19,400	7,566	39	11,058	57	388	2
	2/24/13	105.5	43,600	18,312	42	21,800	50	2,616	6
	2/25/13	103	32,000	15.040	47	13,760	43	1,600	5
19	1/10/13	102	12,000	.....	..	.....	..	.....	..
	1/11/13	102.5	7,500	3,375	45	3,750	50	300	4
	1/12/13	101	5,600	.....	..	.....	..	.....	..
	1/16/13	104.5	10,000	.....	..	.....	..	.....	..
	1/21/13	102.5	9,800	.....	..	.....	..	.....	..
	1/27/13	100.5	14,206	.....	..	.....	..	.....	..
	2/19/13	101.2	25,000	17,500	70	5,500	22	750	3
14	2/17/13	104.5	24,000	12,960	54	8,160	34	1,440	6
	2/18/13	106.5	12,300	6,888	56	3,690	30	861	7
	2/19/13	104	20,600	10,094	49	7,416	36	1,854	9
	2/20/13	102	32,800	19,680	60	9,840	30	2,296	7
	2/21/13	102	24,200	12,584	52	6,776	28	1,936	8
	2/22/13	105	25,300	13,662	54	8,076	32	2,024	8
	2/23/13	102	42,000	27,720	66	10,080	24	2,940	7
2	2/21/13	103	56,200	35,406	63	10,678	19	6,744	12

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White Count						Date of Crisis	Laboratory Examination	Prognosis
Transitionals	Per Cent.	Eosinophils	Per Cent.	Basophils	Per Cent.			
388	2	...	....	...	....			
872	2	...	....	...	....			
1,280	4	...	....	320	1	.....	.....	Died.
.....	..	...	....	...	....	.....	Urine: alb. trace; casts; hyalin; diazo neg. V.P. neg.	
75	1	...	....	...	....			
.....	..	...	....	...	....			
.....	..	...	....	...	....	Lysis.		
.....	..	...	....	...	....			
.....	..	...	....	...	....	.....		
750	3	500	2	...	....	.....	.....	Cured.
480	2	960	4	...	....	.....	Urine: alb. +; casts: hyaline and granular.	
492	4	246	2	123	1			
1,030	5	206	1	...	....			
984	3	...	....	...	....			
2,904	12	...	....	...	....			
1,518	6	...	....	...	....			
1,260	3	...	....	...	....	.....	.....	Died.
3,372	6	...	....	...	....	.....	Urine: alb. trace. Aspirated fluid;	

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the crisis; in one, three days before; in two, two days before; in one, one day before; in three, on the day of the crisis; in two, two days after, and in one, three days after the crisis. In three complicated cases none reappeared in the counts made.

In only one instance, Case 21, did the lymphocyte curve cross the neutrophil curve.

SUMMARY OF SERIES 5, TABLE 7

1. Bronchopneumonia in Series 5 was either secondary in poorly-nourished infants or followed by complications. Three died and one recovered.

2. The total leukocyte counts varied with the associated findings and complications.

3. There was a constant tendency to high leukocyte counts.

4. Eosinophilia was present in two cases and absent in the other two cases.

SUMMARY OF TABLE 8

1. High total white count usually following an earlier decrease after crisis or lysis.

2. High neutrophil count and absence of eosinophils.

SUMMARY OF TABLE 9

1. None of the cases with serous effusions showed much over 20,000 total counts, and one was as low as 11,600. The neutrophils were constantly high.

2. The large lymphocytes varied, but there was no tendency toward a lymphocytosis, although all gave positive tuberculin tests, and the eosinophils were absent in all cases at the time of examination.

SUMMARY OF TABLE 10

1. High total leukocyte count.

2. Low neutrophil and high small lymphocyte count.

3. Absence of eosinophils.

SUMMARY OF TABLE 11

1. If pus infection precedes the crisis, the total white and neutrophil counts remain high.

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2. If infection becomes manifest following the crisis and the count has dropped, there reappears an absolute neutrophil increase.

3. With secondary infection, eosinophils usually disappear, if present, following the crisis.

SUMMARY OF TABLE 12

1. Eosinophils were slow in disappearing during the early part of the illness and returned the day following the crisis in normal numbers.

A case to compare with the one in my series showing worms is Professor Lichtenstern's case of ankylostomiasis. In 1897, the percentage of eosinophils in the blood was 72. The patient acquired croupous pneumonia during the acme of which the number of eosinophils dropped to 6 or 7 per cent., then rose to 54 per cent. during convalescence from the pneumonia. After the removal of the worms the number fell at once to 11 per cent. In 1898, when the ankylostomata were few and Charcot crystals were no longer present in the feces, the number of eosinophils amounted to 7 per cent.

Arneth, in a series of cases examined for eosinophilia during the course of pneumonia, arrives at the following conclusions:

Eosinophils were present in 50 per cent. of eleven cases examined during the course of the pneumonia. Fifty per cent. of these showed eosinophilis, in very limited numbers, however. They were cases of very short courses and abortive cases and in one a chronic case (the only one of his series). He concludes that the severer the case the more certain are we of a complete disappearance of eosinophils. Shortly after the chill we may find an occasional one. They usually reappear (seven cases) on the day following the crisis, in one case before the crisis. In nine cases there was a post pneumonic eosinophilia; in six cases there was no increase above normal, in most of the former the normal per cent. was only occasionally passed.

Arneth believes that eosinophils play no rôle in the fight against the bacterial invasion or the development of the antibodies in pneumonia. He also believes that Türk must change his view that the eosinophils, as do the leukocytes, play an important rôle in the fight against the invasion. Arneth bases his belief on the continued absence of eosinophilia in some cases or its late appearance long after the crisis in others. The presence of eosinophilia is very varied in different cases. From his one chronic case in which the eosinophils persisted throughout the course,

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he raises the question as to whether they do not in time become accustomed to the infection, this explaining their constant presence (one case seems too little to judge), but my Case 41, in which there had been two previous attacks, gives practically the same findings. A need of them results in their recurrence in greater or less numbers. Arneth does not believe that their return is due to chemotactic influences. The above series of Arneth were aged 42, 28, 58, 64, 44, (?), 37, 54, 33, 18 and 10 years, respectively.

Naegeli states that eosinophils are absent or rarely present until near the time of the crisis. Often the first ones are found one or two days before the crisis. They increase more rapidly after the temperature drops until we actually have a post infectious eosinophilia. In itself this is a sign of good omen as it indicates that the intoxication is decreasing.

Japha states that in the postfebrile period of pneumonia there is frequently found an increase of eosinophils, and sometimes an outspoken eosinophilia, though as a rule the increase is moderate. From a clinical standpoint, all that has been learned is that the ordinary metabolic products of bacteria repel the eosinophil cells.

Türk believes that the eosinophils in bacterial infections play a rôle directly opposite that of the neutrophils. They disappear entirely from the blood-picture or are present in very small numbers. In croupous pneumonia they usually return again shortly before the crisis. During convalescence a distinct and absolute increase can often be demonstrated, although this rarely reaches a high degree. An early and marked increase in pneumonia frequently indicates an abortive type. Türk further states that large mononuclear leukocytes and transitionals run parallel to the polymorphonuclear neutrophils and there is often an absolute increase and they remain high even after the crisis. Myelocytes and megaloblasts may occur in severe cases.

It is of interest to note that a peculiar relation seems to exist between the appearance in the blood and tissues of lymphocytes and eosinophil cells; neither are apt to appear when the neutrophilic cells constitute the prevailing cell in the blood or exudate, but either may be found in the less acute exudative processes. The sequence of lymphocytosis followed by eosinophilia is especially clear after the injection of tuberculin (Grawitz), and after splenectomy in animals (Kurloff). In the majority of cases reported as showing lymphocytosis no distinction is made between

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lymphocytes and large mononuclear leukocytes, yet there is considerable evidence to show, as Erhlich believes, that these cells represent a separate theory and in some conditions the large mononuclear leukocytes and not the lymphocytes are increased. In Von Jaksch's anemia, rachitis (Rieder), syphilis (Rille), scarlet fever and small-pox and acute leukemia (Frankel), the significance of the increase of the large mononuclear cells, apart from the lymphocytes, cannot at present be stated.

CONCLUSIONS AS TO PNEUMONIA

1. Leukocytosis in pneumonia as in other infections is dependent on two great factors: first, the reaction on the part of the organism, and second, the severity of the infection. This is peculiarly true of children whose course in the typical types is associated with a lower mortality than in adults while in the atypical, secondary types, we see the highest mortality of any age except that of senility.

2. Generally speaking, a high count indicates a severe infection in an individual of strong powers, a moderate increase indicates either a slight infection with good resistance, or a severe type with an inadequate reaction. A low count suggests either a slight infection or an overpowering influence.

3. It is impossible to make a reliable prognosis in pneumonia from the leukocyte count alone; however, when studied in relation to the clinical picture, the leukocyte count becomes of great prognostic significance.

4. Primary pneumonias of infancy are usually of the lobar type. Bronchopneumonias are usually either secondary pneumonias or are seen in infants with low vitality. This assertion is based on the physical findings, clinical course and blood-findings.

5. Young infants react to infections with a high neutrophil count, the same as seen in older children.

6. The usual blood-picture in pneumococcus infections is that of a high neutrophil count with a moderate lymphocytosis before the crisis. A deviation from this picture should lead one to suspect lues, tuberculosis, typhoid or influenzal or other infections, or the presence of a rachitis or other complication. Excessively high counts are apparently more prone to the development of empyema and other complications, although it is impossible to say whether or not the hyperleukocytosis is

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not the result of an already infected pleura, etc., not demonstrable clinically. In several instances the toxic symptoms associated with these high counts have led us to do spinal puncture with negative findings.

7. The neutrophil and lymphocyte curves approximate and occasionally cross each other during convalescence. There is a rapid fall in the neutrophils after the crisis.

8. Pseudocrisis is associated with little or no reduction in the total count.

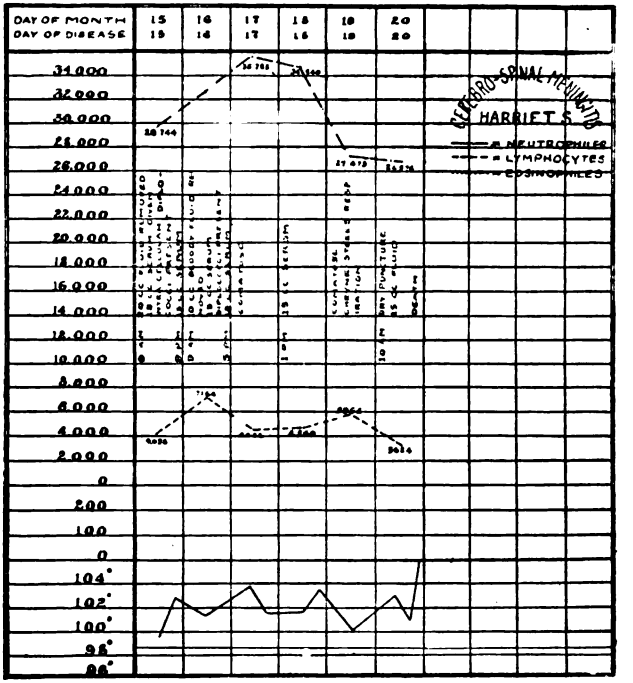

Chart VII—Case 6.

9. A slow fall in the leukocytes with continued high fever indicates either delayed resolution or complications. This is commonly seen in the secondary pneumonias.

10. Sudden increases in the total and neutrophil counts during convalescence should lead to the suspicion of complications, as should a tendency to remain high.

11. In secondary pneumonias the count varies with the associated complications. Frequently there is a high lymphocyte count.

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12. Eosinophilia:

1. Eosinophils practically disappear during the height of the disease and usually reappear just before or following the crisis or with the beginning of lysis. Their recurrence can usually be considered a good sign.

2. Disappearance of eosinophils does not depend on the presence or absence of a leukocytosis. When they have disappeared, however, the absolute decrease tends to remain until the white count nears the normal.

3. They rarely reach an absolute count much above the normal.

4. Their occurrence usually indicates that the infection has passed its acme, and is therefore of practical value for prognosis.

5. Their disappearance may be retarded by outside factors, as the presence of ascaris lumbricoides in the intestinal tract in Case 50 and repeated pneumococcus infections in Case 13.

6. Their disappearance following their return with beginning convalescence should lead to the suspicion of beginning complications.

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CERTIFIED MILK

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The propaganda for clean milk has been one of the chief factors in the development of the "Social Welfare" movement during late years. In all our centers of population certified milk is an important item in the food supply of the baby and its production engages the attention of many of our best sanitarians and pediatrists.

We find that there is still prevalent in the profession, even where we would least expect it, much misconception as to what certified milk is and what benefit is to be expected from its use. Thus we often hear certified milk spoken of as if it were identical with pasteurized milk, and I have known physicians to advise against the former on the ground that to heat milk made it unfit for infant-feeding, although certified milk is, of course, not heated at all. In THE JOURNAL of the American Medical Association we find the question asked, "Is Certified Milk Safe?"[1] and the emphasis is there laid on the dangers which are inherent in all milks and against which even certified milk cannot absolutely guard. In the *American Journal of Diseases of Children* we find quoted[2] without unfavorable criticism a standard of 30,000 bacteria per cubic centimeter; while in a paper[3] read before this Section last year it is asserted that a "waste of money and effort accompanied the work of many of the charities

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3. Cooley, Thomas B.: Relation of the Infant-Welfare Movement to Pediatrics, THE JOURNAL A. M. A., Dec. 2, 1912.

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which confined their efforts to the distribution of clean milk, of which so much was once expected, but which accomplished little more than to help to prove that impure milk plays by no means the most important part in the causation of infant mortality." What the writer of the article doubtless had in mind was the now generally recognized truth that impure milk is but one of the many factors in our tremendous infant morbidity and mortality, and that measures for the prevention of summer diarrhea and similar diseases, which are predicated only on the furnishing of clean milk for children's use, have not been so successful as at first was anticipated; but that these efforts were barren of results as affirmed by the writer of the criticism, is absurd. All agencies intended to improve the living conditions of the children of our poor take their origin from the pure milk movement, and have, as one of their most important functions, the proper feeding of the babies under their control. This means, as a primal necessity, that the milk furnished them shall be clean.

Because of lack of information on this subject among the profession at large, and because of the recent adoption by the American Association of Milk Commissions of a code of requirements which it is hoped will eliminate discrepancies in the requirements of different commissions and which will make the production of certified milk uniform throughout the country, it has seemed to the officers of the association that this was an opportune time to bring to notice the provisions of this code and to make more accurate the general conception as to what certified milk is.

The certified milk movement originated with Dr. Henry L. Coit,[4] who, after several years' ineffectual effort to improve the milk supply of his community through legislation and other ordinary means, devised the plan for a commission consisting of medical men, which, by

4. Dr. Coit's writings are freely quoted without definite reference.

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voluntary supervision and control of methods, by paid expert inspection of dairy work, and by their certification of the product, should undertake, in cooperation with dairymen possessing the requisite intelligence and capital, to make available a supply of milk produced under regulations imposed by themselves, which should fulfil the most exacting requirements of the physician.

Under the leadership of Dr. Coit, the first medical milk commission was organized in 1893, in Essex County, New Jersey. The term "certified milk" was coined to designate the milk produced under the control of this commission, and was protected by United States copyright. In addition to this, many states have since passed statutes prohibiting the use of the term except on the authority of a properly constituted commission. The movement, originally designed by its author to meet the dire necessities of his own neighborhood, was so successful that similar commissions were organized elsewhere—in New York in 1896, in Philadelphia in 1897, and since then throughout the country—until there is now a total of not less than sixty commissions in twenty-three states, and two in Canada. New York leads with ten, Ohio has six, while California in the extreme West and Massachusetts in the East each have five. The American Association of Milk Commissions was organized in 1906, to institute better methods of propaganda and to unify the work of its component commissions. In California there is also a state association which aims to keep the work within its jurisdiction on the same high plane.

The influence of these commissions has been so profound, not only within the limits of their own immediate activities, but in relation to the improvement of the general milk-supply and all other food products, that they may be classed as one of our most important agencies in public health work; and Dr. Coit, through whose continued ardent advocacy of the cause of pure milk thousands of babies have been saved from needless sick-

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ness and death, may properly be named among our great medical philanthropists.

The first step in the acquisition of a supply of certified milk in any community is the organization of a milk commission. This should consist of about five physicians, selected by the county medical society solely because of their known interest in all that the pure milk movement stands for, and perhaps a public-spirited attorney or one or two other lay members should be added to it. Their term of office should be five years, for experience in the work is essential for useful service on a commission, and the personnel should be changed as little as possible. The members receive no pay, and the expense of the work, the fees of the experts, printing, etc., are provided for by a system of assessments on the dairies certified.

An approved and trustworthy dairyman, with financial ability and adequate dairy facilities must then be found, who will agree, in consideration of medical support and an increased recompense in the price of his product, to conduct his dairy, collect and handle the milk in conformity with the requirements of the commission.

Formerly, each commission fixed its own requirements and naturally there resulted a great variety of standards. The code of the American Association was adopted last year only after the most painstaking consideration of its provisions by a committee of able and experienced members, and it may be held to represent the most authoritative conception of the conditions under which milk should be produced for certification. Membership in the association will hereafter depend on acceptance of and compliance with this code, and it is hoped that the heretofore heterogeneous standards of the various commissions will soon be obsolete.

The duties of the members of the commission shall be to enforce the regulations, which, in the code referred to, cover in minute detail every phase in the production of certified milk; to be responsible for personal inspection of the dairy and distributing stations at such inter-

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vals as will serve to keep them thoroughly in touch with the management; to provide for competent expert examination of the herd by approved veterinarians, and bacteriologic and chemical analyses of the milk at proper intervals; to maintain such supervision over all matters pertaining to the health of employees as will protect the milk against contamination by pathogenic organisms from those engaged in handling it; and to advance the doctrine of pure milk by private precept, by public lectures, exhibits, literature and all other available means.

The control of the commission over the dairy and the entire process of production and distribution of the milk must be absolute. The hygiene of the dairy is the first consideration. The terrain of the farm must be such that it is well drained and the paddocks and yards shall be kept clean and dry. Rubbish and stable dirt shall not be allowed to accumulate nor shall any insanitary condition be permitted. The water-supply must be pure and ample.

The dairy buildings must be located at a sufficient distance from other buildings, toilets or latrines. dusty roads, cultivated and dusty fields, or other possible sources of contamination. If unavoidably exposed to dust the buildings must be screened with cheese cloth.

The stables must be so constructed as to facilitate the prompt and easy removal of waste products. The floors must be made of non-absorbent material, must be properly graded and drained and have cement gutters at least six inches deep to catch and carry off all manure and drainage. The walls and ceilings should be planned to avoid lodgment of dust and dirt. At least six hundred cubic feet of airspace should be allowed for each cow, with not less than four square feet of window area. The buildings must be screened to prevent the entrance of flies, rats, and other vermin. The greatest cleanliness must be maintained about the premises at all times, the stables are to be washed out twice daily, and all feed and water-troughs kept clean and sanitary.

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The proper selection of cows for a certified herd is a matter of importance, a proper admixture of animals of different breeds being frequently necessary to maintain the requisite fat standard. The productivity of each cow in a herd should be recorded and the unprofitable ones weeded out, as a few such in a herd lower its average output very appreciably and entail financial loss to the owner. No cow should be admitted to a certified herd until its freedom from tuberculosis or other disease has been determined by the veterinary expert of the commission, and the entire herd must be subjected semi-annually to a rigid tuberculin test, which shall conform to the rules and regulations of the United States government with respect to tuberculin testing.[5] Veterinary supervision must be maintained at all times and sick cows at once removed from the herd and quarantined or destroyed as the nature of the illness may require. In California there is what should be regarded as a valuable rule in relation to the exclusion of tuberculosis from the herds, namely, when a group of animals from one source is examined for admission to the herd, if more than 10 per cent. react to tuberculin, the entire group is rejected. We do not believe it safe to admit to a certified dairy even non-reactors from herds known to be extensively infected, as this will almost surely permit animals in the incipient stage of the disease to gain entrance. Each animal is identified by a numbered ear-tag or other mark, and a descriptive record is kept of distinctive markings, dates, results of tuberculin tests, etc.

Cows should be kept clipped and before each milking must be thoroughly cleansed by brushing and washing, the udders especially being washed clean and dried. All utensils used in handling the milk must be sterilized, and the milkers must wear clean washable suits and carefully cleanse their hands before milking. One of our Western dairies under probationary certification during

5. Circular of instructions for making tuberculin tests, U. S. Bureau of Animal Industry.

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the past year, has been using milking-machines with which great difficulty was at first encountered in keeping down the bacterial counts, but this is apparently being obviated by experience and minute attention to detail.[6]

The milk must be strained, cooled and bottled immediately after being drawn and the bottles sealed with a cap bearing the name of the dairy and of the certifying commission, together with the date of production or of permissible delivery. The bottles must then be packed in ice in suitable containers for shipment and must reach the consumer within thirty hours, the temperature at the time of delivery not to exceed 50 F.

Chemical and bacteriologic examinations of the milk are to be made at least weekly, and in the event of any deviation from normal standards, tests shall be made daily until the discrepancy has been corrected. If this cannot be accomplished within ten days, certification shall be withdrawn. So delicately balanced may be the organization of a certified dairy and its success so dependent on perfection of technic, that I have known the sudden loss of a cleaner to be at once discovered by the bacteriologist, the added work thrown on the remaining hands and the slight relaxation in methods having resulted in an immediate rise in bacterial content.

The methods that must be followed in carrying out the chemical investigations essential to the protection of certified milk are so complicated that, in order to keep the fees of the chemist within moderate limits, those procedures must be eliminated which are not strictly necessary to demonstrate the quality of the milk. For this reason the determination of the water, the total solids and the milk-sugar is not required as a part of the routine examination by the code of the American Association.

6. Since writing this paper, certification of this dairy has been canceled.

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The fat standard is fixed at 4 per cent., with a permissible range of variation of from 3.5 to 4.5 per cent., while the standard for certified cream is 18 per cent., with an allowed variation of 2 per cent. in either direction. The tests recommended are the Babcock, the Leffmann-Beam, and the Gerber.

The protein standard is fixed at 3.5 per cent., with a permissible range of from 3 to 4 per cent., to be determined by the Kjeldahl method, the Gunning or other reliable modification being used, and the factor 6.25 being employed in reckoning the protein from the nitrogen. The protein determination may be omitted except when some special condition renders it desirable.

The chemical examination also includes tests for adding coloring matter and preservatives, among the latter being formaldehyd, borax, boric, salicylic and benzoic acids, and salts of the latter; and tests to determine if the milk has been heated. The specific gravity should be between 1.029 and 1.034.

The bacteriologic standard of the American Association is not to exceed 10,000 bacteria per cubic centimeter of milk, and the presence of pathogenic bacteria excludes the milk from certification. The samples to be examined must be collected by a representative of the commission, in original packages as offered for sale and they must be kept iced until plated, which shall be not later than forty hours from time of milking. Because of the great variation in methods for the bacteriologic examination of milk and the divergent results thereof, the methods to be followed in the examination of certified milk, the composition of the culture mediums, the technic of plating and the period of incubation are all carefully specified, thus affording a proper basis of comparison between milks from different sources. The experts of the commission are required to render written reports of each examination made by them, these to be kept on file in the office of the secretary.

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The milk laboratory is a development of the pure milk industry which now, in a number of our cities and in connection with certified dairies, affords the physician an opportunity to order for his little patients milk formulas much more accurately compounded than is possible by home modification, which is a very great advantage in infant-feeding.

The conditions under which certified milk is produced make it impossible that it shall ever constitute a large part of the total milk-supply. As long as we continue to use milk in its natural state, however, we may feel sure that no higher grade of milk can be obtained and that certification will continue to exert an ever-increasing good influence. The one disadvantage urged against it is that of its higher cost in comparison with ordinary milk. This is apparently a valid objection, but it may be stated that in no other combination of foods obtainable in the markets can the same food value be obtained for the price which certified milk commands. The objection, therefore, is more apparent than real.

Chief among its advantages are its known and constant chemical composition and nutritive value; its relative freedom from bacteria of any sort and its practically absolute freedom from pathogenic organisms; its early delivery to the consumer in sealed and iced containers; its purity and freedom from adulteration; and its consequent adaptability to the dietetic requirements of children and invalids.

That these qualities are assured by the guarantee of physicians actuated solely by philanthropic motives in their milk commission activities, merits for certified milk the confidence of the profession and the public. We feel, therefore, that we may expect the unqualified support, especially of pediatriasts, and of all those who are interested in the public health.

ABSTRACT OF DISCUSSION

Dr. J. W. Van Derslice, Chicago: I am not a Holstein man, I am a Brown-Swiss man. In the pure-bred Holstein it

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is practically impossible to run a uniform fat percentage. There is no animal in which atmospheric changes, fright, etc., affect the contents of the milk so much as in the Holstein. Last fall we had a severe thunder-storm; the following day the fat of the Holstein farms dropped to 2.6 per cent. The thunder-storm did not last over fifteen minutes.

Another thing to be considered is the milk-machines. We had one farm with milk-machines and we did not have any trouble with the bacteria count. After we had educated the milkmen to the certified-milk plan they turned out milk of very low bacterial count. We did find that veterinary examination of that herd was important. It was necessary to examine the herd weekly, and practically every time it was necessary to throw out one or more animals on account of mastitis. So the milk-machine was discredited. I hope that next year I can tell you that the milk-machine is the only thing to use. One of the other farms is putting in milk-machines, and we hope that improvements have been made in this machine so that it will not develop mastitis. If we could get machines that would not cause disorder of the udders, the question of the production of sanitary milk would be answered.

Dr. Henry L. Coit, Newark, N. J.: In connection with the initial steps made to secure certified milk, I wish to say that unaided I could never have accomplished it. Without the cooperation of the producer my work in the beginning would have been negatived. Had it not been for the loyal cooperation of physicians my plans would have failed. The name of the dairyman who produced the first quart of certified milk should be recorded in history. It is Stephen Francisco. Without the practical dairyman, the medical milk commission could accomplish little.

Had it not been for the loyal support of the representative medical milk commissions this association would not have been founded. Geier, Freeman, Hamill, Tuley and Myers gave the impetus in founding this movement. I wish simply to supplement the kindly reference to me by mentioning the names of others who did constructive work.

Dr. John Zahorsky, St. Louis: In St. Louis we have met great difficulty in keeping the bacteria count down to 10,000 because of the hot weather. Generally we have it down even below this, but in spite of the same care it will run up. The commission has insisted on uniform standards. I still think that there will have to be some variation in standard in order to meet climatic conditions. So far as the effect on the infant is concerned, it does not matter whether the bacteria count is 10,000 or 30,000. There will have to be a minimum and a maximum count, and not a definite fixed line.

Dr. Thomas C. McCleave, Berkeley, Cal.: I do not believe that a dairy which produces milk only under 30,000 has any right to ask for certification by milk commission in any part

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of this country. The work of North in New York state is probably familiar to you all. We know that he teaches that, under almost any conditions of environment, almost any intelligent dairyman can produce milk of a lower bacterial count than 10,000. In my cities the ordinary market milk is crowding certified milk very closely in the matter of bacteria count, and for us to have on the market in any city a certified milk in which we insist that this count come down to only 30,000 would be most mortifying, when you consider that in that city ordinary market grades of milk may be found to have lower bacterial counts than certified milk. Any dairyman who is not willing to produce his milk under conditions which will guarantee an average of 10,000 or less should not produce certified milk. We have no difficulty in keeping the count below 10,000. Our dairymen themselves would be mortified if we suggested that they come down only to 30,000. The matter of uniformity of standards is, of course, a debatable question. The object of the American Association of Milk Commissions, as I understand it, is to assure to physicians who are not members of the association of milk commissions and the public that when they ask for certified milk in New York or Brooklyn, or Chicago, or San Francisco, or anywhere they know what they are getting. The bacterial count, the care of cows for protection against tuberculosis and all the requirements should be the same. It is even true in this country that there are commissions putting the stamp of guarantee on milk from cows which have not been tested for tuberculosis. The whole propaganda of the milk commissions is discredited by one commission of this kind. We do not expect that every commission will be able to come up to our standards absolutely. In various parts of the country climatic or other conditions may make it impossible to do this; but all we ask is that the commission come up to the standard to the best of its ability. If we have an honest commission the result will be fairly good. When commissions are willing to give their names to something concerning which they know nothing, then we have a product which is misbranded and should come under the ban of the pure food laws. Some members of commissions do not know what certified milk is. That is the reason I wrote this paper—there is so much ifinorance as to what certified milk is. In some parts of the country the laity are much better informed along this line than the physicians.

Mr. Stephen Francisco, Fairfield, N. J.: Our plant does not produce certified milk only. The idea is to make certified milk safe for infant feeding, and I pity the producer who does not consider infant feeding above everything else. It takes more than the cattle there to produce the milk desired. The herd has to be examined every day. There is hardly a day that some cow is not taken from our herd for one reason or another,

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generally not to return. We are learning something every day, through the suggestions of our physicians, bacteriologists, chemists and veterinarians—every one of whom is giving thought to this matter. Certified milk costs between 12 and 13 cents a quart.

MR. W. E. MILLER, Lebanon, Ohio: The cost of certified milk may be estimated in percentages as follows: Wages, 28.26; supplies, 2.40; stable hauling, .31; power and refrigerations, .58; repairs, 1.31; freight, 5.46; feed, 47.23; cost of certification, 8.42; miscellaneous, 4.79; bedding, 1.24.

I have taken the cost of milk from our books for 1912. This covers one year, and I have taken the total cost of all the items that should enter into the problem and have reduced them to percentages.

There is no uniform system of accounting among the producers of certified milk. Each man must take into account local conditions. It is rather hard to compare the results of two men. Perhaps this can be worked out in the near future. If we can arrive at a uniform way of accounting it will help to clear up the cost of prodluction. We know that in some places the production of certified milk is a fad. Men with more money than they need select this method to get rid of it. It seems to me that philanthropy and business do not mix well. If this movement for the production of certified milk is to succeed, it must be put on a sound economic basis. We do not advance far in any business unless the firm profits, and these profits should include a proper compensation for everyone concerned. I have heard of certified milk selling for from 12 to 20 cents a quart. If the milk is the same all over the United States as it should be, then why this difference in price? Some men say that they cannot afford to sell certified milk for 12 or 16 cents. Perhaps there is something in their organization that needs improvement. We should first be sure that we have approximated 100 per cent. efficiency and then, if the price of certified milk is not sufficient to give a fair compensation, it is time to ask for an increase in price. All fair-minded producers are trying to reduce the cost as much as possible. We have organized our force into three divisions —a cleaning division, a feeding division and a milking division, in order to get 100 per cent. efficiency.

The cost of certification, including the cap as well as the inspection, is 8.42 per cent. I think that the consumer should not be burdened with the unnecessary cost of supervision and capping. We should try to secure trustworthy men, so that the milk need not be kept under lock and key. We must, of course, protect the public as much as possible. After the public is protected our supervision should stop.

Another thing that increases the cost of certified milk is the appearance. We have to take into account the psychology of the situation. Things have to look clean as well as be

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clean. Can anybody tell me in what respect a white suit is any cleaner than a blue suit or a black suit? It simply looks cleaner. If we are to spread this gospel of certified milk among the consumers, among physicians, we must appeal to the eye. The bacteria count may be good, but the people cannot see the germs; they can see the color of the suit, which may have nothing whatever to do with the bacteria count. That is one thing that keeps up the cost of production. The thing that appeals to me is the advantage of bringing certified milk within the reach of the greatest possible number.

These cost items do not include distribution. The milk is in Cincinnati, the freight is paid, but the milk has not yet been distributed to the consumer. This certified milk is distributed with the other milk. It is, therefore, impossible for us to distinguish the cost of distributing certified from other products. If we had nothing but certified milk it would be easier.

DR. OTTO GEIER, Cincinnati: Will you analyze the cost of certification?

MR. MILLER: It includes cap and inspection by the veterinarian, inspection by the bacteriologist and chemist and all charges of that nature.

DR. J. W. VAN DERSLICE, Chicago: What is the price per pint?

MR. MILLER: Four and three-quarters cents. Wages and repairs and freight are all higher on certified milk than they are on common milk, and feed also is higher.

DR. T. C. MCCLEAVE, Berkeley, Cal.: Mr. Miller has not answered the question adequately. A large part of the cost of certified milk, as compared with ordinary milk, is dependent on the addition of the actual expense of certification. It depends also on the greatly increased running expenses of the dairy. For instance, the matter of putting in steam sterilizing plants and sterilizing every utensil that is used, requiring men to wear clean suits, which have to be provided and laundered. The cost of tuberculin testing and the cost of the animals destroyed when found to be tuberculous are large items. All those items enter into the question over and above what Mr. Miller specified. If a dairyman started to produce an equal grade with certified milk, irrespective of any relation to the commission, his expenses leaving out fees, would be higher than those of the man who produces in the ordinary way.

DR. J. W. VAN DERSLICE, Chicago: In Chicago the milk is produced practically under two contracts. Under one the man furnishes the bottle, label and certification and sends the milk f. o. b. to Chicago for 12 cents. The distributor orders a certain number of bottles each day. The other contract, which brings the cost of certification and the cost of distribution more clearly before you, is, I believe, the best one. There are four or five firms under contract to distributors, who

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agree to take the entire output of the firm at 8 cents a quart net, the distributor furnishing the labels, paying the cost of certification and buying the bottles. On that 8-cent basis, the farmer—and the farmer I am speaking of knows how to figure costs—says he is getting $2 a hundred for common milk. In other words, he says the difference between the production of certified milk and the production of commercial milk is that, if he sells his milk as a commercial milk, with the lessened cost which that entails, he would sell it on a graduated scale running from $1 to $1.70 per hundred. For the year 1911 the average was $1.67. For the year 1912 it was a little higher. This man says that he is selling his commercial milk for $2 a hundred. In other words, the men who figure the cost for us most closely tell us that there is a leeway of 40 cents per hundred pounds, or 40 cents per 46 quarts, between the production of certified and commercial milk. This philanthropy then is a fine text for a sermon. They do not talk that to me any more. The men who went into this business first have their money tied up in a big plant. They knew nothing about what the possibilities were for them, but the man who goes into the business to-day does so as a money-making proposition, because there is a market for the product. The rich man wants some place to take his fine friends, and the only possible way to make the farm pay is by the sale of certified milk. There is absolutely no question about it. The big farm to-day that is not selling its entire output is losing money. According to the government reports, over 50 per cent. of the farms are losing money to the owner every day.

Another point I wish to bring out is the value of the superintendent. We can show you superintendents who have changed from one farm to another, and the bacteria count follows the superintendent. You must have a man who knows, feels and breathes certified milk. Another point Mr. Francisco spoke of—in one-half or two-thirds of our farms a prize is held up to the employees on the farm—a bonus of $5 a month if the bacteria count during the month is under 5,000. In that way you have every worker on the farm watching his coworker to see that there is no carelessness. Every man wants that $5.

Dr. Otto P. Geier, Cincinnati: There is nothing about which there is so much confusion as the cost of certification. I attempted several years ago to make a survey of the methods used by the different commissions of the country and to define in my own mind, at least, the broad problem of the cost of certification. There are just as many methods of arriving at the cost of certification as there are milk commissions in the country. One commission charges a per bottle tax and supplies nothing but the chemical and bacterial work and veterinary inspections. The next one charges a flat rate for each bac-

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teriologic chemical and veterinary examination, and the producer buys his caps. In still another instance the producer pays a per bottle tax and the chemical, bacteriologic and veterinary inspections are made by the commission. The commission also buys the caps and seals out of the per bottle tax income. So you have just as many variations in the matter of arriving at the cost of certification as you have commissions. I have not seen the figures shown here before. At first sight they seem unreasonably high. I should remark, however, in the interest of Cincinnati that the commission does many things for the producer. It supplies the caps and seals, medical inspections, veterinary inspections and chemical and bacteriologic examination, which are done biweekly, and more frequently if there is any trouble at the dairy. The commission is able to maintain a laboratory for this work in connection with the office of the commission. The bacteriologist and chemist and his assistant, as well as the medical profession of the community, are at the call of the dairymen. The commission also does propaganda work, thus advertising the product of the dairymen and in a general way serving the community from a broader point of view. Everything else being equal, it is fairer to use a per bottle tax than a tax which applies only to the various examinations. In a per bottle tax the large producer helps pay the load of the smaller producer.

Mr. W. E. Miller, Lebanon, Ohio: The cost of keeping up the herd is based on the assumption that the herd is practically stationary. Individuals go out, but others come in. We have practically no loss from the tuberculin test. As a matter of fact, the herd increased in value during the time under discussion. As to repairs, our aim is to keep the equipment in the original condition. It is practically as valuable now, because of improvements made from time to time, as it was in the beginning.

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SPASMOPHILIA WITH ESPECIAL REFERENCE TO FAMILIAL REACTIONS AND REPEATED ABSENCES

JULIUS PARKER SEDGWICK, M.D.
MINNEAPOLIS

New light has been thrown on the subject of convulsions and allied conditions in childhood by the study of the electrical reactions. This work was begun and carried out for years by Escherich.[1] General convulsions in children, laryngospasm, spasmodic apnea, tetany and similar phenomena have been shown to be but manifestations of an underlying hyperexcitablity of the peripheral nervous system, with a tendency to tonic and clonic spasms, the so-called spasmophilic diathesis.

Mechanically the hyperirritability can be shown by tapping over the seventh nerve along its course in the cheek, with a resulting contraction of the muscles around the mouth. Tapping over the upper branches of the same nerve may produce a similar contraction about the eye. The first is known as Chvostek's and the second as Weiss' phenomenon. Constriction of the arm in this condition at times brings on the tetany position of the hand—Trousseau's phenomenon. The Chvostek and Weiss phenomena are therefore indicative of spasmophilia. The Trosseau phenomenon is pathognomonic.

More important, however, because they can be more accurately measured, are the electric reactions. In the normal child, with the Stinzing electrode applied over the peroneal nerve, no cathodal or anodal opening contract-

1. Escherich, Theodor: Die Tetanie, Hölder, Vienna and Leipsic, 1909.

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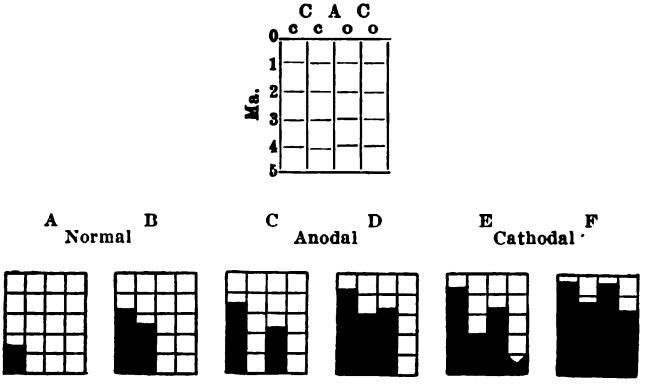
tion is produced with less than five milliamperes of galvanic current. Charts 1 to 6, as given by von Pirquet,[2] show the normal reactions as well as those of hyperexcitablility.

The first column, C C C, represents cathodal closing contraction, the others in order, A C C, anodal closing contraction, A O C, anodal opening contraction and the fourth, C O C, cathodal opening contraction. The amount of current necessary to produce the contractions is shown by the height of the blocking indicated by the number of milliamperes on the left of the standard specimen blank chart above charts 1 to 6.



Charts 1-2.—Normal reactions given by von Pirquet.
Charts 3-6.—Hyperexcitability reactions given by von Pirquet.

The study of the electrical reactions has shown that there may be a latent spasmophilic condition or an active state with such manifestations as have been mentioned above. Ibrahim states that at certain times of the year 30 per cent. or more of the children show spasmophilia latent or active. The importance of the recognition of this latent condition, in order that the proper prophylactic treatment for the prevention of convulsions may be instituted, is evident.

2. Cited from Escherich: Die Tetanie.

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Characteristic pathologic anatomic changes have not been shown in the nervous system of spasmophilics. Escherich, basing his conclusions on the work of Yanase and Erdheim, considered the condition due to changes in the parathyroids. Later, negative findings by various investigators have, however, thrown doubt on this hypothesis.

The studies of Quest and others on the brains of infants indicate a low calcium content in these cases. Mac Callum and others have demonstrated that the calcium of the brains of animals suffering with tetany parathyreopriva is low. Cohn and Cooke have on the other hand, had somewhat contradictory results. Herman Schwartz[3] suggests that these conflicting results may mean that it is the calcium-sodium ratio which is of most importance as in the case of this character in which the calcium content was high the sodium content was also high, making in one of Aschenheim's cases a quotient of forty-six instead of the normal one from nineteen to twenty-six. Although Gerstenberger did not succeed in obtaining improvement in these cases by the oral administration of calcium lactate and Grulee's metabolism work gives little or no confirmation of this hypothesis, it is very probable that there is some relationship between spasmophilia and the calcium metabolism.

Manifestations of spasmophilia are much more common in the late winter and early spring months. Breast-fed children are rarely affected. According to Finkelstein[4] the whey of cow's milk is an exciting factor. Infectious diseases or alimentary disturbances may change a latent spasmophilia into an active condition with convulsions. Rickets and spasmophilia frequently occur together.

It has long been recognized that this condition is hereditary or familial. The literature, however, shows

3. Schwarz, Herman, and Bass, Murry H.: The Calcium Metabolism in Infantile Tetany, with Report of a Case, Am. Jour. Dis. Child., January, 1912, p. 15.

4. Finkelstein: Lehrbuch der Säuglingskrankheiten, Berlin, 1905, Part 1, p. 248.

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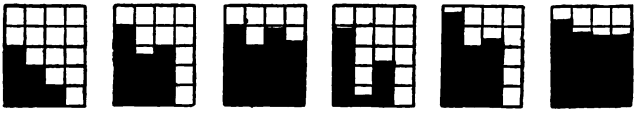
few records of familial electric reactions. Schiffer[5] reports one family in which the mother and one child of 15 years gave normal electric reactions, and two of the children showed hyperelectric excitability.

Toyofuku[6] took the electric reactions of four newly born puppies whose mother had been subjected to the removal of the thyroid and parathyroid glands. All four showed marked tetany reactions. The mother showed definite signs of tetany and heightened electric reactions after the birth of the puppies. He considers the condition as a lactation tetany. He believes that a tetany or tendency to tetany may be transmitted through the milk.



7 8 9 10 11 12

Charts 7 and 8.—John D., Case 1, April 13 and April 18, 1911.
Chart 9.—The nursing mother of John D., Case 1, July 25, 1911.
Charts 10 and 11.—Anne B., Case 2, Nov. 22 and Nov. 26, 1912.
Chart 12.—The nursing mother of Anne B., Case 2, Nov. 22, 1912.

Without knowledge of this work on dogs I have examined the electric reactions of the mothers of the somewhat rare cases of spasmophilia in breast-fed children which have come to my attention, and have found the irritability of the mothers almost uniformly high.

CASE 1.—John D. was 5½ months old when first seen by me, March, 1911. He was still at the breast. His aunt had had convulsions as a child and she is now mentally deficient. The mother stated that she took four quarts of milk and cream daily during her pregnancy and at the time of the examination her diet was almost wholly made up of milk and cream. The infant was brought to me because it had had repeated convulsions. The child was markedly rachitic with pronounced craniotabes. His electrical reactions were spasmophilic as shown in Chart 7 (April 13) and Chart 8 (July 18).

The markedly spasmophilic reactions of the nursing mother of John D. are given in Chart 9 (July 25).

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5. Schiffer: Ueber familiare chronische Tetanie, Jahrb. f. Kinderh., 1911, lxxiii, 601.
6. Toyofuku, T.: Beitrag zum experimentellen Studium der Lactationstetanie, Jikazasshi M. 130, 1911; Cited from Ztschr. f. Kinderh., 1911, i. R., p. 592.

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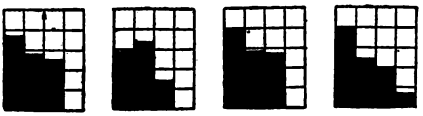
CASE 2.—Anne B. was 5 months old and at the breast at the time of her first examination. She had rickets with marked craniotabes and gave the reactions shown in Chart 10 (November 22) and Chart 11 (November 26).

The nursing mother gave a marked Chvostek phenomenon and her electrical reactions were striking, as shown in Chart 12 (November 22).

CASE 3.—William R. was 9 months old and at the breast when he came under my care, Nov. 14, 1912. He had had repeated convulsions from his fourth month. He still has convulsions and there is a decided mental injury. His reactions are shown in Chart 13 (November 15), and Chart 14 (December 15).

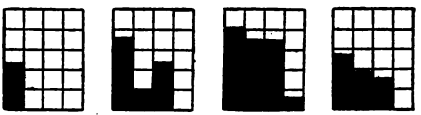


13 14 15 16

Charts 13 and 14.—William R., Case 3, Nov. 15 and Dec. 15, 1912. Charts 15 and 16.—The nursing mother of William R., Case 3, Nov. 15 and Nov. 20, 1912.

The nursing mother's reactions show marked hyperexcitability (Chart 15) November 15, and Chart 16 (November 20).

CASE 4.—E. G. a girl of 3 months, and at the breast, had convulsions, but no demonstrable hyperelectrical excitability. (Chart 17), September 4. She had been given chloral hydrate.



17 18 19 20

Chart 17.—E. G., Case 4, Sept. 4, 1912. Chart 18.—The nursing mother of E. G., Case 4, Sept. 4, 1912.
Chart 19.—The nursing mother of George L., Case 5, May 29, 1912.
Chart 20.—The nursing mother of Irma E., Case 6, October 29, 1912.

The nursing mother, however, gave evidence of hyperexcitability on the same day. (Chart 18), September 4.

CASE 5.—George L. died at the age of 1 year with bronchopneumonia and beginning tuberculous meningitis following pertussis. There was marked tetany for some time before death. Unfortunately, the child's reactions could not be taken, but the reactions of the mother, who was nursing him, are shown in Chart 19 (May 29).

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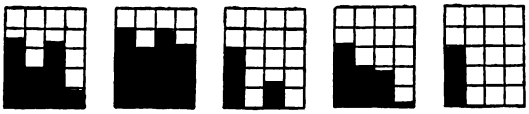
Case 6.—Irma E. was under my care at the age of 6 months with pyelocystitis. Although her electrical reactions were not taken, she had marked tetany of both hands. The nursing mother's reactions are given in Chart 20 (October 29).

In but two cases of spasmophilia which I have seen in infants at the breast, the mothers have not shown very marked electric excitability. One of these children showed marked carpopedal spasm, but was getting other food as it was in its third year. The other was also older, 2 years and 3 months, and also had marked tetany.

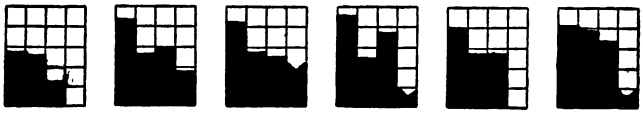
To recapitulate, in all of the cases of infants 1 year or less of age at the breast whom I have examined and found spasmophilic, the nursing mothers have shown definitely heightened electric excitability.



21 22 23 24 25

Chart 21.—John D., Case 7, Aug. 1, 1911. Chart 22.—The mother of John D., Case 7, July 25, 1911. Chart 23.—The brother of John D., Case 7, Nov. 21, 1912. Chart 24.—The aunt of John D., Case 7, Oct. 22, 1912. Chart 25.—The grandmother of John D., Case 7.

The electric reactions of other members of the families of spasmophiliacs often bear out the frequently made assertion that the condition is hereditary or familial.



26 27 28 29 30 31

Chart 26.—Baby R. R., Case 8, Feb. 13, 1913. Chart 27.—The mother of Baby R. R., Case 8, Dec. 26, 1912.
Chart 28.—Baby Q. T., Case 9, Dec. 28, 1912. Chart 29.—The mother of Baby Q. T., Case 9, Dec. 28, 1912.
Chart 30.—Theo. McC., Case 10, March 21, 1913. Chart 31.—The mother of Theo. McC., Case 10, March 21, 1913.

Case 7.—In the family of Baby J. D., as shown by Chart 21 (Aug. 1), the mother, brother and aunt have heightened electric reactions, as well as Chvostek phenomena, Chart 22 (July 25), Chart 23 (November 21) and Chart 24 (October 22). The aunt had had convulsions as a child and is now mentally

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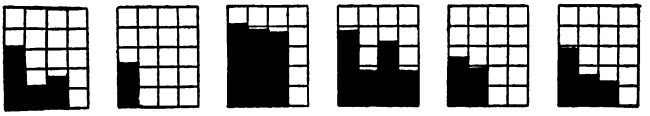
deficient. An uncle had had convulsions during his first year. The grandmother presented a marked Chvostek phenomenon, but without heightened electrical excitability. (Chart 25).

CASE 8.—Baby R. R. with convulsions and a marked Chvostek phenomenon gave spasmophilic reactions. (Chart 26, February 13). The mother's reactions are shown in Chart 27 (December 26).

CASE 9.—Baby Q. T. had convulsions and marked spasmophilic reactions. (Chart 28, December 28.)

The mother had had convulsions as a child and the high reactions shown in Chart 29 (December 28).

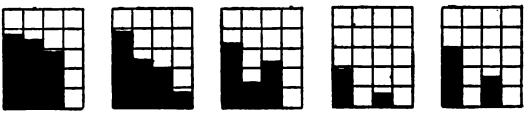
CASE 10.—Theo. McC. has had convulsions, and his mother gave similar reactions. (Charts 30 and 31, March 21.)



32 33 34 35 36 37

Chart 32.—Baby B. J., Case 11, July 27, 1912. Chart 33.—Mrs. J., the mother, Case 11. Chart 34.—Mr. J., the father, Case 11. Chart 35.—Virginia J., Case 12, April 3, 1913. Chart 36.—The mother of Virginia J., Case 12, March 1, 1913. Chart 37.—The father of Virginia J., Case 12, Nov. 1, 1912.

CASE 11.—Baby B. J. gave spasmophilic reactions (Chart 32, July 27), the mother did not, but the father did (Charts 33 and 34). All three showed the Chvostek phenomenon, the father's being especially marked.



38 39 40 41 42

Chart 38.—William R., Case 3, Nov. 20, 1912. Chart 39.—The mother of William R., Case 3, Nov. 20, 1912. Chart 40.—The father of William R., Case 3, Nov. 21, 1912. Chart 41.—The paternal grandmother of William R., Case 3, Nov. 20, 1912. Chart 42.—The paternal grandfather, Case 3, Nov. 20, 1912.

CASE 12.—This is also true of Virginia J. (Chart 35, April 3), her mother (Chart 36, March 1), and father (Chart 37, November 1).

CASE 3.—Baby W. R., had active spasmophilia for months with reactions shown in Chart 38 (November 20).

Mrs. R., his mother, showed marked hyperexcitability (Chart 39, November 20). His father (Chart 40, November 21), as well as his parents, the grandmother and grandfather (Charts 41 and 42, November 20), showed less marked evidences of hyperexcitability.

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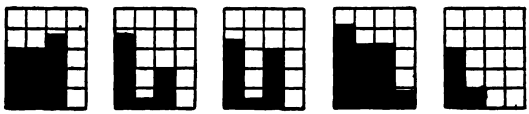
Case 13.—The B. family in which the father had had convulsions as a child, consisted of nine children, eight of whom had had convulsions in childhood. The three who could be examined gave the heightened reactions shown in Charts 43-45, together with the Chvostek phenomenon. The last reaction is interesting as it is that of the only son who did not have convulsions, showing a latent spasmophilia.

Further repetition from my records would only consume time. The cases in which one or both parents of spasmophilic children do not give like reactions are exceptional.

We are indebted to Friedmann for the recognition of the character of the repeated non-epileptic "absences" in children, which so resemble petit mal. He considers that they bear no relation to epilepsy and apparently



43 44 45 46 47

Chart 43.—Mr. B., Case 13. Chart 44.—The son of Mr. B., Case 13. Chart 45.—The only son of Mr. B. who did not have convulsions.
Charts 46 and 47.—S. S., Case 14, Aug. 8 and Sept. 12, 1912.

have nothing to do with hysteria. They are characterized by the following peculiarities: They begin in apparently well children from the fourth to the seventh year, usually suddenly after some excitement, such as fear or operation. They consist of short, light disturbances of consciousness without convulsions and without falling. They come on at first in a great number of attacks (6 to 10 to 100 a day and occasionally at night). The course of the disease is tedious, usually being from seven to eight years. There may be also long remissions. They have no disturbing influence on the mental or bodily development of the child. This is a chief argument against their relation to epilepsy from which they, moreover, differ, in that the occurrence of convulsive attacks is almost never observed (*fast völlige Ausbleiben*). They are also to be distinguished from epilepsy by the history.

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Against their hysteric nature, the uniformity of the attacks, in spite of continuance for years as well as the lack of other symptoms which would make one suspicious of hysteria, may be cited. The "repeated absences" present a good prognosis in that they appear to clear up before puberty even without treatment. So far as the systematic placing is concerned, they may be related to the narcolepsy of Gelineaus and the other somewhat half-understood "neurotic abscences" of adults. It seems that they appear on the foundation of a spasmophilic diathesis as, in the three cases which have been so far recently tested electrically, the increase of electric excitability and cathodal opening contraction with less than 5 milliamperes has been observed (two of these observations were made by Mann).

CASE 14.—S. S., aged 7, seen first Aug. 3, 1912. She has been under my observation during the past year and illustrates the above condition as described by Friedmann. Her father and mother are living and well. She was a full-term child and the birth was normal. She has had no acute infections and although she lived two years in the Philippines she did not contract malaria. Her father was in the habit of giving her as many as six drinks of whisky a day, and the mother thinks that the "spells" which she has had have followed the abuse of alcohol.

Two years ago she began to have attacks which were diagnosed as petit mal. She usually had from one to fifty of them and has had as high as one hundred a day. The attacks were first noticed at the table. She would stop eating and stare off into space. During the attacks she does not answer when spoken to. The first attacks lasted only a few seconds each, but at the present time they are sometimes much longer. She was examined by two very competent men who said it was epilepsy. They gave her bromids and advised country life and all the milk and eggs she could take. She was taken to the country and given the medicine prescribed, without improvement. While in the country she would sink to the ground when the attacks came on and at times had as many as one hundred "spells" a day.

The physical examination showed a fairly well-nourished, gracile child of dark complexion. She made quick movements which were not of choreiform character. The general physical examination showed nothing of importance. The electrical reactions, which were markedly spasmophilic, are shown in Chart 46 (August 3), and Chart 47 (September 12).

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On August 3, during the examination in the office, she looked off into space and the pupils became somewhat dilated and the expression blank. The attack lasted about ten seconds, after which she immediately became the active, alert, apparently normal child she was before. She was sitting on the edge of a table from which she did not fall and seemed to have no difficulty in keeping her position. She said nothing during the attack, although she had been talking on and off before. During the attack she seemed to notice that her dress was unbuttoned in the back, although the expression was still vacant. There was no Chvostek phenomenon. The patellar reflexes were very marked. The Achilles tendon reflexes were present and marked.

She was put on a carbohydrate diet without milk or meat and given phosphorus in cod-liver oil, but no sedatives.

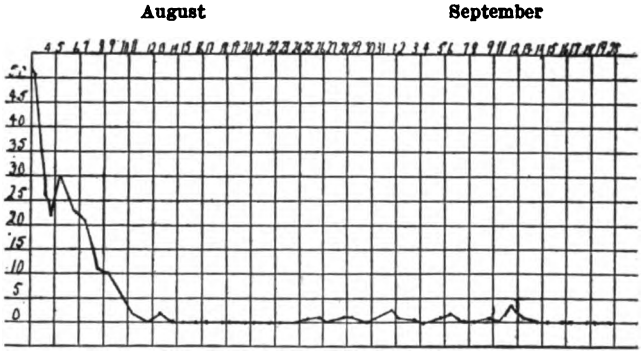

Chart 48.—Decrease in epileptic attacks. Case 14, Aug. 4 to Sept. 25, 1912.

The decrease in the number of attacks can be readily seen from the curve extending from August 4 to September 25, 1912. (Chart 48.) On September 12 she left Minneapolis, but continued with the milk-and meat free diet and the mother reported from St. Joseph, Missouri, on Oct. 26, 1912, that she was having about one attack a week and that very slight. The treatment was continued. On Nov. 26, 1912, she reported from Cheyenne, Wyoming, that the child had had no attacks since October 26. The treatment was ordered continued. On Dec. 20, 1912, she reported from Tacoma, Washington, that the child had not had any more attacks and was doing well. During the spring of 1913 she had a recurrence, but she was in the West and there was no opportunity to take her reactions. As the spasmophilic season of the year passed over she improved.

Case 15.—Another case, but with some motor symptoms, which is still under observation with slight improvement is that of Edith N., aged 11. The mother's brother is said to

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have had epilepsy at the age of 4 or 5 years, but as he recovered and is now well he may have had spasmophilia. This child began to have attacks two years ago. She was sitting on a table during examination. Without any warning the eyelids dropped, the eyeballs showed nystagmus and turned upward slightly. As she leaned forward there seemed to be an attempt to right herself as one who falls asleep in a chair. The attack lasted from three to five seconds. She seemed normal immediately afterward, but could give no proper description of the attack. She has from twenty to thirty-five of these daily. The electrical reactions may be seen in Chart 49 (January 10). If this case is epilepsy, there is spasmophilia present also, as shown by the electrical reactions.

It is possible that the following case which shows both petit mal like attacks and convulsions may be a link between "repeated absences" and the spasmophilic convulsions. In my opinion, such electric reactions and the Chvostek sign warrant a better prognosis than in epilepsy proper.

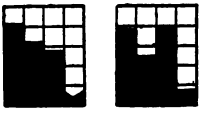

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Chart 49.—Edith N., Case 15, Jan. 10, 1913.
Chart 50.—Joe D., Case 16, Sept. 12, 1912.

CASE 16.—Joe D. was a well-nourished boy of 9 years when first seen in September, 1912. His testicles have not descended. His infant brother was seen by me in typical spasmophilic convulsions and now shows marked anodal hyperirritability. One year before the first examination Joe D. had two or three attacks a day for one week. In these attacks he fell and was rigid and unconscious. The attacks then ceased until five days before this examination. He has had from two to six attacks daily since. His mother says that they manifest themselves at this time as a blank expression which comes over his face for a few seconds. He answers questions at this time but has no recollection of them afterward. During the physical examination he had just such a manifestation as his mother described. He appeared perfectly normal immediately afterward. His electric reactions are shown in Chart 50 (September 12). The Chvostek phenomenon was pronounced. These attacks continued, with some which showed clonic convulsions, until October 10. From that time until

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the present, June 4, 1913, he has had no more seizures. The history with the electrical reactions and Chvostek phenomenon warrant the diagnosis of spasmophilia.

The diagnosis of spasmophilia rests on the electric reactions, convulsions, tetany, laryngospasm, spasmodic apnea, together with the Trousseau, Chvostek and allied phenomena. Not only have we here an aid in the differentiation of these convulsions from those of major epilepsy, but certain cases of petit mal may belong under this heading.

Although death does occur in the general convulsions and in laryngospasm, under proper treatment the prognosis, so far as life is concerned, is usually favorable. Thiemich and Mann found, however, that about one-third of these cases show later evidences of injury to the mentality.

The accepted treatment consists in the withdrawal of cow's milk with the substitution of breast-milk when possible, or carbohydrates in the shape of gruels, and the administration of cod-liver oil and phosphorus. Sedatives, such as chloral hydrate, may be necessary in the active stages. The calcium therapy is not as yet generally used. The recent studies in this subject will require rewriting of the chapters on convulsions to be found in most of the texts on pediatrics.

Syndicate Building.

ABSTRACT OF DISCUSSION

DR. A. C. ROGERS, Faribault, Minn.: I should like to ask what percentage of cases show familial reactions.

DR. MARY STRONG, Omaha: I would suggest that these patients be examined thoroughly as to their mental state by someone who is familiar with psychiatry.

DR. JOHN ZAHORSKY, St. Louis: I should like Dr. Sedgwick to speak more particularly about the treatment. We find great difficulty in withdrawing milk from young babies. We find many children with spasmophilia in infancy, and by gradually withdrawing the milk we improve their nutrition. I have not been withdrawing milk, except temporarily. When it is permanently withdrawn these children may have to be put back on it.

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Dr. Julius P. Sedgwick, Minneapolis: In answer to Dr. Rogers' question, I cannot give the exact percentage, but in almost all cases which I examined the family also showed evidences of hyperexcitability. In regard to the mental state mentioned by Dr. Strong, some patients did show definite mental deficiency. In some of them we used the Binet-Simon test. In regard to the withdrawal of milk, it has been my custom to return these children to the milk diet as rapidly as possible, watching their electrical reactions and thereby guarding against overfeeding.

In older children (I have treated no adults except some of the mothers and they have reported feeling better) I usually reduce the meat diet, because it has been found that meat has some effect. Dr. Zahorsky's position is, I believe, correct

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ROSEOLA INFANTUM

JOHN ZAHORSKY, M.D.
ST. LOUIS

The name "roseola infantilis" had an important place in the medical terminology of writers on skin diseases fifty years ago. Willan distinguished several varieties, and subsequent writers followed his classification with some variations. The older works on pediatrics had descriptions of the disease, but it is obvious from these descriptions that many different eruptions were confused under this heading as Hardaway[1] pointed out. On the advice of the dermatologists the name was dropped and recent text-books on pediatrics do not mention this affection.

For many years I have been interested in a symptom-complex, a febrile erythema, which occurs almost exclusively in infants and which deserves a place outside of the erythema group of skin diseases. To this the older writers gave the name "roseola infantilis," but did not differentiate this disease from rubella and toxic erythemata. In 1909 I discussed this subject before the St. Louis Pediatric society and referred to fifteen cases which were seen in private practice.[2] I wish to repeat some of the remarks made previously and to report twenty-nine additional cases (Cases 4 to 33).

The patient is almost always a child under 3 years of age who suddenly becomes ill with a high fever. The physician is called and on an examination of the patient finds nothing to account for the fever. The fever con-

1. Hardaway: Keating's Cyclopedia of the Diseases of Children, iv, 17.
2. Pediatrics, January, 1910.

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tinues, but no diagnosis can be made on the second, third or even fourth day. Then the temperature drops to normal or nearly so and the child, who has been drowsy and irritable, sits up and commences to play. Coincident with the decline in the temperature a morbilliform rash appears on the face and neck and rapidly spreads over the body. The eruption disappears in twenty-four to forty-eight hours. There are no complications nor sequelae. No desquamation follows the disappearance of the rash.

SYMPTOMATOLOGY

These are striking and characteristic symptoms: A prodromal fever lasts from two to five days, the disappearance of the general symptoms follows the appearance of the rash and the eruption is morbilliform in character. The following brief analysis of the symptoms was given in the previous paper.

Fever.—This is generally high, ranging from 102 to 105. The usual temperature at the physician's visit is about 103. The elevation in temperature is continuous with moderate morning remissions. It is accompanied by extreme restlessness at night and drowsiness during the day. The fever falls by crisis within a few hours after the appearance of the eruption. In one or two cases the fever disappeared a few hours before the eruption was observed. In some cases the eruption covered about one-half of the body.

The Eruption.—This is distinctly morbilliform, that is, a rose-red macular, or maculopapular eruption, showing circular or elliptical lesions varying in diameter from 1 to 3 mm., with healthy skin intervening The rash is most marked on the trunk, less so on the face. The back, buttocks and thighs are generally well covered. The lesions may occasionally be very sparse and limited to the trunk. There is no crescentic arrangement as in rubeola, and the lesions are generally not raised much above the skin.

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The Respiratory Tract.—There are no catarrhal symptoms except congestion of the fauces. Sometimes the disease at the onset may be mistaken for a grippal angina. A macular enanthem was sometimes seen on the palate on the day preceding and coincident with the eruption. Koplik's spots were absent. Slight redness of the conjunctiva was observed a few times.

The Gastro-Enteric Tract.—Vomiting was noted in a few cases. Diarrhea was present a few times. Several of the little patients showed some signs of indigestion, but nothing characteristic.

Desquamation.—None was observed.

The Lymph-Nodes.—The superficial lymph-nodes in the neck were very slightly enlarged in most cases. The axillary and inguinal nodes seemed normal.

ETIOLOGY

All of my cases occurred in infants under 2½ years of age. Sex seems to exert no influence. All but one of the patients were fed on artificial food, or were given supplemental feeding to breast-milk.

The disease is not contagious. Only in one family did more than one case occur. These were two little boys, aged 2½ and 1½ years, who had the eruption three days apart. Older children who have not had rubella remained with the little patients continually without contracting the disease. The comparative isolation of these young children renders the assumption of a contagion almost impossible.

The active cause of the disease is unknown. That it is due to an intestinal intoxication is possible, but there is not much in my series to support this view.

DIFFERENTIAL DIAGNOSIS

There is only one exanthem which need be seriously considered in diagnosis and that is German measles (rubella). Measles, scarlet fever, Duke's disease, and erythema infectiosum have distinctive characters not

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possessed by roseola which are sufficient to separate them positively from the latter disease. So many writers have attempted to separate certain clinical types of rubella from the disease proper that one who will make another attempt is almost subject to ridicule. It is probably true that rubella is a name applied to a group of closely related infections; but roseola has such distinct features that I must insist that it deserves a separate place in our nosology. A parallelism between the two diseases clearly shows the distinction.

In rubella the prodromal fever is but of a few hours duration. The fever rises with the eruption. Children of all ages are susceptible. It is contagious, it occurs mostly in the spring and there is enlargement of the lymph-nodes.

In roseola the prodromal fever lasts three to five days and the fever disappears with the eruption. It occurs mostly in infants. It is not contagious and is prevalent mostly in the fall and winter. There is little enlargement of the lymph-nodes.

Occasionally rubella is characterized by a prolonged prodromal fever, but the other symptoms and its contagiousness separate it from roseola. I have seen rubella in several epidemics and also in isolated cases and yet the syndrome presented by roseola is different.

Roseola cannot be placed in the category of the exudative erythemata for obvious reasons. It is not a drug rash and it differs from the ordinary erythemata in being preceded for several days by fever.

OLDER DESCRIPTIONS

In Meigs and Pepper's text-book[3] the following description is found after describing different forms of the disease, some of which are clearly toxic erythemata: "Again, in a yet more marked form, which frequently but by no means exclusively occurs in warm weather, when it is styled roseola estiva and autumnalis, the erup-

3. Meigs and Pepper: Diseases of Children.

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tion is symptomatic of a more definite constitutional disturbance. It begins with more or less chilliness, alternating with heat, with loss of strength and spirits, with headache, restlessness, sometimes mild delirium, and even, it is said, though we have never seen them, with slight convulsive phenomena. At the same time there is some slight febrile reaction. . . . After these symptoms have continued for two, three, four, or even six or seven days, the eruption appears first on the face and neck, whence it extends in twenty-four or forty-eight hours to the rest of the body. The rash resembles very closely in some cases exactly that of measles; but the catarrhal symptoms are absent. . . . The eruption is sometimes accompanied by itching, and sometimes by stinging pain, and the febrile symptoms generally continue, though moderated in a degree after the appearance of the rash; while in other instances the fever disappears entirely from that moment." I cite this to show that such cases have been observed, although many were confused with rubella, dengue and other eruptions.

Dermatologists probably do not see these cases, as the general practitioner treats them through the febrile stage for a variety of conditions and when the eruption appears there is such amelioration of the symptoms that counsel is not sought.

To show what difficulties in diagnosis are encountered in private practice a history of one of my earlier cases is given.

REPORT OF CASES

CASE 1.—J. C., a boy, 14 months old, had always been healthy. The nourishment was by maternal nursing for seven months from birth, since then cereals and cow's milk have been used with some additional food from the table. There has been no serious digestive disturbance. In May, 1908, the patient was suddenly taken with a high fever (104). The most careful inquiry revealed nothing abnormal beyond a congested throat. As the disease was regarded as an intestinal intoxication, castor oil and restricted diet was prescribed. On the following day the temperature was still high and the mother reported that the child was restless through the night. Again the physical examination revealed nothing. The ear-

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drums were not congested, there was no marked sore throat and no stuffy nose. On the following day a urinary examination for suspected pyelitis yielded no evidence of disease. A blood-count was made—leukocytes 11,500. The blood was also examined for the malarial plasmodia and another specimen of blood was sent to the health department for the purpose of a Widal test. On the fourth day we were still puzzled; but the mother telephoned that the baby seemed better. In the afternoon another message informed us that the patient had an eruption. The rash proved a typical rubella-like efflorescence. A prompt drop in temperature was synchronous with the appearance of the eruption.

During the last three years I have seen about thirty cases of this disease and a brief report of twenty-nine (Cases 4 to 33) is appended.

Of these twenty-nine cases fifteen occurred in girls and fourteen in boys. All the children were under the age of 26 months. Nine cases were seen in 1911, fourteen in 1912, and eight (to June 1) in 1913. Three cases occurred in January, six in February, four in March, three in April, one in May, two in June, one in October, five in November and four in December. July, August, and September were exempt. In this seasonal occurrence roseola resembles the respiratory infections. All the cases occurring during the measles epidemic of 1913 were particularly watched for evidence of measles. Two other cases were excluded from the series because the catarrhal symptoms were marked and the fever did not fall with the appearance of the eruption, although Koplik spots were absent and no history of exposure to measles could be ascertained. During 1912 very few measles cases occurred in St. Louis. During these three years a few cases of German measles were seen in older children, but no general epidemic prevailed.

Gastro-enteric symptoms are rarely prominent. In three cases some diarrhea was noticed. This subsided with the appearance of the rash. One other case seen in June, 1913, was not placed in this series. This was a baby boy, 9 months old, who suffered from diarrhea and slight fever for three days. The stools were frequent

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and acid in reaction. On the fourth day an intense erythematous eruption appeared. It lacked the morbilliform character and was classified with toxic erythemata. I have met this particular syndrome in children above 2½ years of age only twice in the last six years.

Case 2.—G. E., aged 9 years, was seen in August, 1907. The only prominent symptom was an elevation of the temperature (104). Fever with a temperature range of 102 to 104 continued for four days. Every effort was made to make a diagnosis without success. On the fourth day a few roseolar spots were seen on the abdomen and a Widal test was made. On the fifth day the temperature had dropped to normal and the boy felt well. The whole trunk was covered with circular spots resembling the rose spots seen in typhoid. There were no other symptoms.

Case 3..—A. R., aged 14 years, has had measles and German measles. In May, 1913, the patient had an attack of fever, (up to 105), which terminated by crisis on the fourth day and a very profuse rash appeared which resembled rubella. There was no desquamation.

Although this particular syndrome was constantly watched for in older children, these two cases are the only ones seen which can compare with the clinical features as observed in infants. In the years preceding 1911, many such cases were observed, all in infants. It is reasonable to assume, then, that the disease with the clinical syndrome mentioned is almost entirely limited to infants, hence the proposal to call the disease "roseola infantum," a slight change from the older name.

This opportunity is taken to urge physicians to study this symptom-complex. It may prove to be a sporadic occurrence of rubella showing certain peculiarities in infants. At present we must regard it as an interesting clinical symptom-complex which to the practitioner deserves a distinct place.

Case 4.—F. R., a boy, 2 years old, was breast-fed until 5 months of age and was then put on a supplemental feeding of modified cow's milk. There was no serious digestive disturbance until the child was 14 months old, when he suffered from a few attacks of indigestion as solid food was added to the diet. A slight idiosyncrasy against egg was shown at first but was

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soon overcome. On Nov. 20, 1912, the mother noticed that the child had fever. On the following day the temperature *per rectum* registered 104 2/5. During the height of the fever there was an expiratory grunt. The examination of the chest and body revealed nothing abnormal, but a congestion of the fauces was noticed. There was no enlargement of lymph-nodes, no eruption, no enanthem, and no ocular redness. Neither vomiting nor diarrhea had been present. Castor oil and aspirin were prescribed.

On the following day the cervical lymph-nodes were palpable, and the temperature was lower (100). No Koplik spots were present and there was neither eruption nor cough. On November 25, when the patient had been free from fever for ten hours, an eruption was first observed on the trunk. This became very profuse and confluent on the back, but was sparse on the extremities. The rash consisted of rose-red maculopapules. No arm sign was present. The tongue and throat were normal. The lymph-nodes were scarcely palpable. No more fever occurred and the eruption disappeared in two days.

CASE 5.—W. M., a girl, 5 months old, has been fed on diluted cow's milk. A restless and feverish condition existed for three days (March 7, 1911). A profuse morbilliform eruption appeared on the face and body. The temperature was normal. There were no Koplik spots.

CASE 6.—C. H., a girl, 5 months old, is suffering from chronic indigestion and malnutrition. The patient had been fed on Nestlé's food but was placed on pasteurized cow's milk one month before, on which the improvement was marked. Two pounds were gained in one month. (March 20, 1911.) A fever which lasted two days ended with a morbilliform rash.

CASE 7.—E. R., a girl, 20 months old, has been bottle-fed since birth. For a long time a digestive disturbance was present. The child suffered one attack of pyelocystitis. Nov. 17, 1911, fever suddenly appeared (103) No adenopathy, no catarrhal symptoms. It was at first thought to be a recurrence of pyelitis. November 20 typical roseola showed on back and face and a disappearance of the symptoms resulted. Three older sisters had no rash either before or after this illness.

CASE 8.—J. R., a boy, 7 months old, has been nursed by the mother supplemented by three bottles of milk daily, which are composed of ⅔ milk and ⅓ water, with 3 per cent. of milk-sugar added. The weight of the child is 16 pounds, 4 ounces. The last three nights the patient has been in a feverish condition, and is constipated; but had one loose stool yesterday (Nov. 7, 1911). Temperature, 99 2/5. A profuse morbilliform rash covers the trunk. There are no Koplik spots, no ocular congestion, no cough and no enlargement of the lymph-nodes. A sister, 3 years old, had no eruptive disease before or after his illness.

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CASE 9.—J. T., a girl, 9 months old, takes two quarts of certified milk daily. The weight is 19 pounds. Fever has been present for two days, there is a slight cough and a slight discharge from the eyes. The mother gave one dose of a laxative nostrum. There is no fever to-day. There are no Koplik spots, and no enlargement of lymph-nodes. There is a profuse morbilliform rash on the body. An older sister, 3 years old, had no eruption before or after the baby's illness (Feb. 17, 1913).

CASE 10.—N. R., a boy, 8 months old, has been breast-fed entirely since birth. On the last two days fever has been present and several loose stools are passed daily. There is no fever to-day (June 18, 1911), but the body is covered by a morbilliform rash. One eye is slightly congested. The throat is congested, but there is no enlargement of lymph-nodes and no Koplik spots. No medicine has been given.

CASE 11.—A. M., a fine-looking boy, 11 months old, has been fed on dextrinized flour and milk since he was 2 months old. At 9 months some semisolid food was added to his diet. Fever has been present for two days, and the bowel movements are increased. There is no fever to-day (April 1, 1912), and the bowels have not moved. There is a morbilliform rash on the body. There are no enlargements of the lymph-nodes. A brother and sister had no rash before or after the baby's illness.

CASE 12.—E. C., a girl, 25 months old, has had a tendency to eruptions but no idiosyncrasy against any article of food. The patient is a healthy well-developed girl, and is on a general diet. The mother was disturbed about an eruption, Nov. 23, 1912. The child had fever for three days, and two days before the mother ascertained it to be 102. Restlessness at night is marked and waking is with a scream. No gastro-enteric disturbance has been observed. There is no cough. The rash consists of rose-red macular lesions, some elliptiform, others circular, and are most profuse on the trunk. The rectal temperature is normal. A slight sore throat is noticed, there are no Koplik spots, and no enlargement of the lymph-nodes.

CASE 13.—M. W., a girl, 15 months old, is a healthy child, and the mother takes great precautions with the food. There are no other children in the family. The patient is suffering (Dec. 18, 1911) from a morbilliform eruption which is very profuse. There is no fever, and no enlargement of lymph-nodes or enanthem. Four days ago a fretful and feverish condition arose and food was refused. The temperature rose in the night, but the child was playful during the day, though waking several times during last night. The child is very irritable but has no cough, and has received no medicine. The eruption disappeared in three days.

CASE 14.—W. M., a girl, 12 months old, has been bottle-fed since the age of 1 month, and has had repeated attacks of

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indigestion, also once before an erythematous eruption without fever. She is the only child in the family and is kept carefully isolated. Dec. 21, 1912, fever developed and the mother gave castor oil, which evacuated several foul stools. The rectal temperature (Dec. 23, 1912) is 99 2/5 and there is morbilliform eruption on the body. There is no enlargement of the lymph-nodes, and no enanthem.

Case 15.—E. L., a girl, 2 months old, breast-fed, had a mild attack of bronchitis when 1 month old. The child has two sisters and one brother, none of whom has recently suffered from a rash. Fever was present for four days, once rising to 101 4/5, when a morbilliform rash appeared. The fever subsided. There have been some dyspeptic symptoms, the acid dyspepsia of the young infant. Sodium benzoate was prescribed two days before the breaking out of the eruption. Recovery was prompt and no eruptive disease occurred in the family subsequently. (Dec. 5, 1912.)

Case 16.—F. P., a boy, 8 months old, is breast-fed at night, and is given milk and cereal mixture in the day time. There is no digestive disturbance. The temperature on Feb. 14, 1912, was 104. This fever continued three days and dropped to normal suddenly, when the baby broke out with a profuse morbilliform rash. There were no Koplik spots and no evidence of exposure to any eruptive disease.

Case 17.—R. B., a boy, 5½ months old, has been fed on condensed milk; the baby is fat, the weight being 17 pounds. Twenty teaspoonfuls of water and three teaspoonfuls of condensed milk are given every two hours. During the last week there was some constipation. High fever, 103, is present. The conjunctiva is slightly injected. The child is restless. Aspirin and sodium benzoate were given. Feb. 10, 1912, a profuse morbilliform rash appeared. According to the mother's statement, no fever has been present for three days. Neither measles nor German measles was observed in the city at the time.

Case 18.—K. R., a boy, 9 months old, brought to the city from the country. The child is afflicted with cerebral palsy and also suffers from recurrent attacks of bronchitis. (Feb. 27, 1912.) The patient has fever, the temperature rising to 102. Physical signs of bronchitis are present with asthmatic wheezes. Restlessness appears at night. There is no enanthem. Two days later a morbilliform eruption appeared; there was some coryza. The fever disappeared on the day before the eruption and the bronchitis also rapidly improved. Only creosote was given in this case.

Case 19.—E. H., a girl, 6 months old, is fed on cow's milk. The child has had fever for four days and is cross and restless at night. The bowels are somewhat loose. There is no vomiting and no cough. The patient feels better to-day

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(March 28, 1912). There is a profuse morbilliform eruption but no enlargement of the lymph-nodes. There is no other child in the family.

CASE 20.—A. M., a boy, 3 months old, has been fed on the breast, supplemented by a certified milk and water mixture with cane sugar and sodium citrate. The child has been constipated but there is no vomiting. May 19, 1911, the patient began to be restless and feverish. The rectal temperature was 101. Nothing abnormal has been discovered. The baby was put on barley water and breast-feeding. May 25 a morbilliform eruption appeared. Some curds showed in the stools. Half a pound has been lost in weight. There is no fever.

CASE 21.—S. G., a boy, 5 months old, is breast-fed supplemented by cow's milk, diluted. (Dec. 8, 1912.) Fever (103) is present. This continued for three days. On the fourth day a maculopapular eruption appeared, which covered the trunk and extremities. The temperature was 98 4/5. There was slight enlargement of the lymph-nodes. A slight digestive disturbance is present. The baby is breast-fed, but gets in addition three bottles of certified milk modified with water and cane-sugar.

CASE 22.—B. K., a girl, 5 months old, is entirely breast-fed. The child has had fever all night (Feb. 9, 1913). There is no coughing or sneezing. The rectal temperature is 102 3/5; the throat is congested but there is no ear trouble. The fever continued on February 10, with no change in the physical condition. There was no digestive disturbance. February 11, the temperature has subsided. There is a morbilliform rash all over the body and also on the face. There are no Koplik spots. A slight enlargement of lymph-nodes shows. The baby has a sister 3 years old who has not had measles or rubella. The latter had no symptoms of an eruptive disease in the following month.

CASE 23.—A. G., a boy, 1 year old, was fed on malted milk since 3 weeks old, and was also given orange juice and prune juice. The child had an attack of diarrhea at 11 months, when first seen. There is marked evidence of rickets. The food was changed to milk and barley-water with malted milk; also bread and vegetable soup were taken. Improvement was rapid on this food. Three weeks later the boy was suddenly taken ill with a temperature of 105-2/5. (Oct. 15, 1912.) The fever continued for three days, when an eruption appeared on the abdomen. This maculopapular eruption became very profuse in the next twenty-four hours though not much appeared on the face. Confluent dusky-red patches appeared over the buttocks. The temperature became normal October 19. There were no Koplik spots and no catarrhal symptoms. This boy was very sick.

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CASE 24.—B. S., a girl, 10 months old, had severe gastro-enteric intoxication during the past autumn. (Jan. 1, 1912.) The child had high fever for two days. The temperature now is 103 3/5. There are no catarrhal symptoms and no digestive disturbances. The throat is about normal. The ear drums are in normal condition. The diagnosis was very puzzling. (January 2.) The baby is broken out with a macular eruption; the lesions are circular, large and small, and rose-red. The temperature is 100 2/5. There are no Koplik spots and no catarrhal symptoms. The child was well the next day, the rash fading. There was no desquamation.

CASE 25.—J. H., a girl, 1 year old, had been breast-fed until recently but is now on a mixed diet. The bowels were loose three days ago and have been slightly loose since. There has been no vomiting. High fever was present for two nights (102), the fever continuing for two days longer. (April 9, 1911.) Morbilliform eruption has appeared. There is no fever and the pulse is slow. Diarrhea has ceased. The child recovered promptly of all symptoms.

CASE 26.—L. G., a girl, 7 months old, has been fed on a whey and milk mixture. As a young baby the child suffered from severe vomiting, though it looks well now. The patient (Jan. 21, 1912) has had severe catarrhal symptoms for three days—other members of the family have the grip. The baby has had fever (102). A slight diarrhea is present. Roseola has appeared on the body to-day and a rapid defervescence of the symptoms has followed. This baby had several drugs for its "cold." It may have been a symptomatic rash.

CASE 27.—E. H., a girl, 7 months old, was nursed for three months, then fed partially on the bottle—a mixture of milk, water and milk-sugar being given. The child did well on this food. June 10, 1912, the baby began to be restless and feverish, and had one fermented stool. The patient was put on barley-water, June 11. The temperature rose to 103 in the afternoon. There was no diarrhea and no catarrhal symptoms though there was a slight sore throat. June 12, a very profuse eruption appeared which looked like German measles. The temperature is 100. There are no Koplik spots. The baby has lost its drowsiness. A sister, aged 5, had no such eruption before or afterward.

CASE 28.—C. T., a boy, 10 months old, has a sister, aged 5, who had not had rubeola or rubella. The child has been restless and feverish for several nights. To-day (March 28, 1913) he is broken out with a morbilliform rash. There is no fever and no catarrhal symptoms or digestive disturbance. The child has been fed on milk and barley-water.

CASE 29.—A boy, 13 months old, has been fed on some milk and solid food, including potatoes, bread, oatmeal gruel, baked apple, beef juice and cow's milk to which extract of malt has

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been added to prevent constipation. (April 17, 1913.) The patient has had fever for three days which ranged from 101 to 104. There is much restlessness, but no sore throat, no cough and no enlargement of the lymph-nodes. A morbilliform rash has appeared. There is no fever. The recovery was rapid. There are no other children in the family. The child lives in an apartment.

CASE 30.—B. L., a boy, 18 months old, took scarlet fever of moderate severity Feb. 1, 1913. March 4, 1913, when he had recovered almost completely, the temperature rose to 103, and a variable temperature persisted for three days. There was a slight coryza and some cough. A fine roseolar eruption appeared on the body. The temperature was 101. There were no Koplik spots. The temperature was normal on the following day. There is no other child in the family.

CASE 31.—L. M., a boy, 21 months old, has been fed on a mixed diet. Feb. 13, 1913: The fever began four days ago and the child has since been restless. The mother noticed a rash to-day. Maculopapular spots cover the trunk and limbs but few are on the face. There are no Koplik spots and no fever, but there is a slight enlargement of the lymph-nodes. No sore throat is present. The child is suffering also from a frost-bite on the left cheek. There is a younger baby brother. No more fever is shown. There are a number of cases of measles in the neighborhood.

CASE 32.—E. M., a girl, 15 months old, has been on a mixed diet, and is well nourished. The baby lives in an apartment and has had high fever for three days. There is a slight exudate on one tonsil. The child is restless at night. The temperature is normal to-day. A morbilliform rash covers the whole body but there is little on the face. There was a rapid recovery without drugs. She had received two doses of acetphenetidin before the appearance of the eruption. No digestive or catarrhal symptoms were observed. There is only one child in the family (March 28, 1913).

CASE 33.—B. S., a girl, 3 months old, breast-fed, had fever for three days when the rash appeared. The temperature was still 103 when the eruption was noticed. The next day the fever had dropped to 99½. The rash is morbilliform and erythematous. There is intense congestion of the skin. The infant is restless. There are slight catarrhal symptoms. No Koplik spots are present. The stools are frequent and watery (Jan. 12, 1912).

ABSTRACT OF DISCUSSION

DR. F. P. GENGENBACH, Denver: It is important to recognize this eruption, especially in hospital work. I have seen several of these cases in children's hospitals in Denver. Needless to say we were all much alarmed. I think that in most cases the patients do not have such high fever. When they

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do, we think of scarlatina. As Dr. Zahorsky has said, these cases have never proved contagious. I have never seen other cases develop in the hospital after one of these cases. They do frighten us, however, and I think that we should be able to recognize them.

DR. H. W. CHENEY, Chicago: I wish to add to Dr. Zahorsky's statement that I have seen in recent years a number of these cases corresponding to his clinical picture, coming on with this indefinite fever and then a rash. I have been inclined to classify them under rubella or German measles and think that perhaps we had better cling to that term rather than make a new classification which might confuse many who are not careful observers. We know that German measles is not always contagious. Then, too, in German measles there is frequently fever several days before the eruption. I should be inclined to classify these as typical cases of rubella.

DR. JOHN ZAHORSKY, St. Louis: As I mentioned, these cases are usually diagnosed as German measles, but the syndrome is so peculiar and typical that I think it deserves a special place. We might regard it as German measles until we can establish a definite place for it. Its contagiousness has never been demonstrated. German measles and measles are contagious diseases, but these roseola cases are not contagious, so far as my observation goes. These cases show a clinical syndrome so clear and distinct that it should always be borne in mind when trying to make a diagnosis. Therefore, I think that it deserves a place—temporarily at least.

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THE DESIRABILITY OF EARLY DIAGNOSIS OF MENTAL DEFECT IN CHILDREN, AND MENTAL TESTS AS AN AID

A. C. ROGERS, M.D.
FARIBAULT, MINN.

The physician may come in various ways in contact with mental deficiency of varying degrees, and his ability to diagnose a given case will often be taxed severely unless he keeps clearly in mind the essential nature of the condition and brings to his aid all available diagnostic means.

Every physician of experience recognizes the undirected and shifting gaze, the lack of prehensile grasp and the general failure of response to external stimuli through the special senses that indicate idiocy in the infant. He may also recognize the group of symptoms that indicate some form of primary dementia.

He is, however, aware that the great majority of children that show mental abnormality are neither idiotic nor affected with any psychosis and that their condition is not even included in the well-recognized types, usually associated with mental deficiency, such as cretinism, mongolianism, hydrocephalus, microcephalus, etc. He has little assistance from the common textbooks and he must be content to explain the condition in terms of such popular phrases as "a little queer," "rather backward," "not quite like other children," or something equally indefinite. It is not at all uncommon for a physician to tell the parents of a mental defective that the difficulty will pass away at puberty, or to recom-

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mend a course of special training with a view to restoring the child to a normal condition, overlooking entirely the essential feature of the defect.

To reduce the problem to its simplest form let us emphasize the following facts:

1. Mental deficiency is a condition resulting from physical deficiency or impairment in the brain and nervous system which becomes manifest during the developmental periods of infancy and childhood, and is neither curable nor outgrowable, and is not to be confused with forms of pedagogic retardation from remedial causes.

2. It is not, when uncomplicated, to be confused with insanity which represents an alienation from a normal condition usually after mental development has been completed.

3. It may be complicated with a psychosis or epilepsy or other disorder, functional or organic, but when this is the case, the mental retardation or degeneration naturally receives secondary consideration and is usually the result of the disease, or at least, they owe their origin to a common cause.

4. It follows from the foregoing that the diagnosis of mental defect is made by comparing the reactions of the patient to his surroundings with those of the average normal child. When a person experienced in handling mental defectives is called on to diagnose a case, he immediately seeks as complete a knowledge of the history of the activities of the child as may be necessary to make the comparison clear; the less the mental defect, the more complete the history required; the reactions in the home, on the playground and in the school form the basis for diagnosis rather than symptoms observed by casual direct examination.

Thus the problem, as ordinarily presented to the physician—and it is usually the family physican who first discovers it, or is first consulted concerning the child that is "not right" mentally—is a psychologic one. Whether or not any definite pathologic condition might

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previously have been discernible, it goes without saying that in the great majority of cases of this kind, as presented to the physician, no definite lesion can be discovered that accounts for the mental condition—and it is the mental condition that causes his advice to be sought—and so he is at a loss for any standard by which to make his own mental picture of the case clear, to say nothing of meeting the anxious inquiries of parents and friends with a satisfactory diagnosis and prognosis, unless there is a profound or near profound idiocy or a marked psychosis. It is here that the system of measuring intelligence comes into service, and the Binet-Simon tests afford the best means so far devised for this purpose. They are rapidly serving to standardize mental retardation and defect. They represent a system of about fifty-five procedures to which the reactions of the child give the examiner as much information concerning his intelligence as the longer and more indirect methods, which even the expert must otherwise employ. While the system is entirely empirical, it is the selected result of work with normal and abnormal children during a period of twenty years. To better appreciate their *raison d'être* and their application, one should note the following basic principles involved in the child's development.

1. Psychologists teach us that the evolution of the intellect, using the term in the sense of capacity for knowing, is completed in children at about 13 years of age or 15 years as a possible maximum. During the evolutionary period the child is growing in capacity month by month and year by year, more rapidly during infancy and at a lessening rate in later childhood until it ceases at the end of the period stated.

2. It is quite evident from this that if from any cause, whether it be an inherited deficiency in germ cell potency, or toxic or traumatic influence during fetal life, infancy or early childhood, this evolution be interfered with, the child's mental capacity is lessened. The mental

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evolution may cease at an early period—or rather, it may never get under way, as in idiocy; or there may be a simple slowing down so that the child's capacity at any age below 13 is less than that of a normal child at the corresponding age, and its capacity at 13 (or at least 15) never increases. As the system of measuring intelligence enables the qualified examiner to determine the mental age of the child, it is obvious that if its development be retarded, the amount of retardation will be expressed by the difference between the mental age and the chronologic age, if the child is under 13, or 13 less the mental age if over 13.

As to reliability of tests it is unnecessary here to say more than that while there is considerable difference of opinion in regard to the relative value and proper age adaptation of certain particular tests, these are minor details. Goddard and Kuhlmann, who have done more work with the tests in this country than any other psychologists, as well as others, like Huey, Wallen, Towne, Terman and Childs, who have done enough to speak with authority, agree as to their general reliability.

As to the qualifications of an examiner, when the tests were introduced into this country by Goddard, there was considerable apprehension in regard to their use by any but expert psychologists. The fact is now conceded that this is not justified. I was at first inclined to share this view, but after observing the working of the system as applied methodically and thoroughly to over 1,500 mental defectives and on over 1,000 schoolchildren (by Kuhlmann and under his direction), and after observing the work of different teachers under training in the use of the system, I have become convinced that this is unnecessary. What is more to the point, however, Goddard, Kuhlmann and other psychologists do not consider it necessary that the examiner be a trained psychologist. He should, however, have the psychologic attitude toward the child, and should be properly trained in the use of the tests and how to evaluate the reactions

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to them. Some people make good examiners and others do not, with the same training. Women, other things being equal, make the best. The situation is somewhat analogous to that of the constructing engineer and the operating engineer of a locomotive. The mechanism is invented and perfected by the psychologists and they have given full instructions as to its use. The examiner must learn the technic and must have the right temperament, especially the faculty of getting the full confidence of the child and securing full and free response. The psychologists can be depended on to remedy any defects discovered in the mechanism.

Already the system is being applied in many public schools, especially in the large cities, either as a regular test for special cases or for making surveys. So much for the system of measuring intelligence.

The physician, naturally conservative, not inclined to be carried away by new "fads," may properly inquire:

1. Why should our profession employ a system of measuring intelligence which at most only deals with "symptoms."

2. Assuming that it will be of service in determining intellectual levels, how is the result to be translated into definite diagnosis or prognosis; in other words, what amount of retardation indicates mental defect?

The symptoms—the functioning of a partially developed mind—are exactly what the physician is after at that point in the history of any given case when the mental tests would be called into requisition. The arresting cause, whether it be a lack of hereditary force in the germ plasm, chemical poison, sepsis, or traumatism, has done its work and the physician is studying the patient as an imperfect human product; hence, the problem is, what is to be done for this patient? What training and development is he capable of? We all know that normal children are capable of learning to act, to think, and to inhibit action, with increasing effectiveness as they increase in age and experience, and,

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inversely, the amount expected of a child will be proportionately less as its age approximates birth. If, then, the child passes the age of 13 with a markedly retarded mental development, this is recognized as a defect and the intellectual level at which the development stopped will in general determine the educational capacity of the child and will be expressed by his mental age, or the age of a normal child at the time when this ability is found. Thus, these "symptoms," while not pointing to any definite cause or pathologic condition, as might be the case in a type of fever, are an index of the condition that must be known to suggest the treatment and training to be employed.

The next question, as to how much retardation constitutes mental defect, while not answerable with scientific accuracy, does not leave a margin of uncertainty that invalidates the general usefulness of the examinations. Suppose we say arbitrarily that three years' retardation at 13 indicates positive mental defect, this will leave a safe margin, as less retardation usually represents incompetence. The examiner can make a very workable table for practical use based on this assumption, and intended to apply to lower ages. Suppose the retardation be three years at 13, then we may assume for practical purposes that $^{3}/_{13}$ is the constant factor of retardation. At 9 years the retardation would be approximately 2 $^{1}/_{13}$ years and at 6 years, 1 $^{5}/_{13}$ years, etc. If a greater retardation is found, it simply means the use of a correspondingly larger factor and a lower mental age.

Now it is not to be understood that this means of determining the retardation at any other age than the one at which the examination is made is scientifically correct. It is only a ready method of making an approximate estimate. Psychologists have not worked out the problem of the rate of the evolution or development of mind in normal children. That is, how it varies in uniformity from birth to 13, nor is it yet ascertained

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whether there is a definite relation between the normal rate and the retardation rate in mental defectives.

Again, we would not expect that mental retardation from traumatic and possibly other postnatal causes, would follow any rule, because the cortical areas or nerve lines affected would obviously vary greatly. As 60 per cent. to 70 per cent. of all cases of mental defect are probably hereditary, the field for the uses of the tests is not greatly limited by this fact, and they are useful, obviously in acquired cases. Some of these cases in which the cause is traumatic, will show a sudden and complete arrest of development at the time of the accident.

Now as the higher faculties are the last to develop, it is obvious in theory and verified by experience that with one or two years' retardation even, there is usually poor judgment, lack of forethought, weak inhibition in relation to appetites, and in general, lack of capacity and resistance to cope with the social and economic conditions of life, although capable of doing useful work under the guidance of a higher intelligence. With slight retardation, fair success depends entirely on the environment. Education, of course, is to be determined by the intellectual level found to exist and expressed in terms of mental age—temperament or adaptability to different occupations being considered, as with normals. It is the capacity for manual or routine employment, as a result of training that confuses the uninformed as to the nature of mental defect, because the kind of work mental defectives do so well—and which has resulted from education of the existing capacity—has not called for the exercise of higher control faculties that would be necessary in a successful life of independence. It must be remembered that after all the real test is ability to succeed in life; laboratory tests are only to determine whether or not the faculties essential to insure proper reactions to the natural conditions of life are there and available. The person's variations in temperament or

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reactions to various interests must be reckoned with. For example, the average mental age of fifty boys in one of our farm colonies at Fairbault is 6.9. The highest mental age represented is 11. This latter "boy" is 46 years old and can do any kind of work under general supervision required of a farm hand. He has no initiative, and when an assignment has been completed he will sit down and wait for another. He happens to be of a quick temper and he will resent orders from any one but the head of the department. He will milk six or seven cows and do it well, but he is not a good teamster, largely by reason of his temper.

There are two "boys" each with a mental age of 3; one aged 51, does a little work sweeping walks and doing errands, though he cannot talk and of course cannot read, so must carry notes as messages. He will do his sweeping regularly without being told. Of course the whole amount of work done is but little, though it is a sort of an epitome of his whole life. The other 3-year mental-age case can do good work directly under supervision, in digging post-holes and loading posts on wagons, etc., yet if not watched, will walk off aimlessly. A peculiarity about him in the hayfield is that he can spread the hay after the mower, but cannot pitch it.

One "boy," mental age 4, 26 years old, milks six cows night and morning and does it well. He recognizes the time to start by the sound of the signal bell and gets his pails and starts without further instructions. This is the only thing he has learned to do independently, and he is neat and clean.

One "boy," mental age 6, aged 32, handles an ox-team entirely alone, feeding, hitching, driving and caring for them. He does whatever is assigned with a team without direct oversight and sticks to it until completed. He is interested in his work and will go and finish, uninstructed, work left incomplete by any of his comrades.

Most of the teamsters who handle horses on wagons and farm tools are of 6, 7 and 8 years mental age—

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steady but shiftless, and needing considerable supervision.

Thus defectives of the same mental age react quite differently to the same occupations.

From this it will be better understood that three years' retardation is a safe margin for the average physician to use in diagnosis.

So far, we have considered intellectual levels only. Now, there is another class of cases of mental defect that, strangely enough, are quite often recognized as mentally defective, though the test shows them to be normal intellectually. They do not react normally to their environment, even when this is excellent, either from lack of power to inhibit emotion and action, from lack of ethical conception, or lack of initiative and ambition. There seems to be a lack of coordination between the intellectual and the emotional phases of the mind. As they come to us for examination, the first question raised in each case is, Has this child had a good early training? In many cases the children are orphans and there is good reason to infer that their training has not been good; shifted as they have been from place to place and deprived of an affectionate and sympathetic parental influence, the emotional nature has not always had a healthy and normal development. Notwithstanding this, there are the other cases, by far the most numerous, that under the same environment develop normally, suggesting the probability that there was something fundamentally wrong in the mental makeup of some, at least, of these children. Then there is the occasional case that has had the very best environment and training, including excellent discipline, and yet makes an absolute failure of life and becomes a wreck and even outcast. It seems therefore—these children all developing to be mentally defective—that we have ample justification for recognizing at least one other group of mental defectives than the one before discussed, which

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is characterized by low intellectual levels that can be determined by the Binet-Simon tests.

This leads us to the subject of classification of defectives, generally, and a word as to the use of the terms. In 1910 the American Association for the Study of the Feeble-Minded, recognizing the importance of the long-neglected standardization of terms, and also recognizing the fact that the application of the system of measuring intelligence to which Goddard had called attention the previous year, had not only made it possible to classify on the basis of mental age, but also would doubtless lead to other differentiations, adopted tentatively, only, a classification of intellectual grades, as follows:

Feeble-minded	Moron	Mental age, 8 to 12
	Imbecile	Mental age, 3 to 7
	Idiot	Mental age, 0 to 2

The whole question of the "moral imbecile," "defective delinquent," and other special and emotive types, was set aside for further consideration.

It seems to me that we are now approaching the time when specific groups must be recognized of those who fail to conform, through developmental defect other than that of the intellect. The psychologic scheme could be completed by naming each group by the defective mental quality, to which non-conformity is due, as "moral deficiency"—lack of ethical conception, "inhibitive deficiency"—lack of power to control one's actions, etc.

It should be noted also that the terms "feeble-mindedness" and "mental deficiency" are equivalent and general. The morons are the higher group of intellectual defectives that can earn their own living under proper conditions; imbeciles, the intermediate group that cannot earn their own living, but can protect themselves from common physical dangers; idiots, the lowest group, that cannot protect themselves from common physical dangers. The term "retardation" is used in two senses,

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(1) in school work where there is a failure to keep up with the grade, or pedagogic retardation, depending on remedial causes, and, (2) in cases of actual mental defect.

In conclusion, the purpose of this paper is to urge on the physicians the adoption of the principle of measuring intelligence in all cases of abnormal mentality in children as a part of routine practice. The desirability of early diagnosis of mental deficiency is self-evident, (1) for the information of the physician that he may advise intelligently, (2) for the sake of the parents, who should be wisely guided in their handling of the child, that they may not expect too much and yet may do the practical thing, which is the most economical, and, (3) in the interest of the child, (*a*) that he may neither be neglected at the time when much can be done, nor forced like a hot-house plant to struggle for years in efforts to do things beyond his capacity, and, (*b*) that his limitations may receive protection from the influences that would tend to criminality.

There is no reason now why the physician should not have available a good examiner and make use of this clinical assistance. He can in this way confirm his diagnosis of mental deficiency or disprove the condition, as the case may be, in a large number of cases. The doubtful cases will thus be reduced to a minimum. The Binet-Simon tests, in the hands of a qualified examiner, are practical for the purpose of determining intellectual levels, and no one need hesitate in making them available because of improvements that may be found necessary thereafter, or certain limitations that characterize them in minor details.

Bear in mind that no laboratory method of diagnosing emotive defects is yet available, or, in other words, any method of determining in advance whether or not a child who passes the intellectual tests will succeed in life under ordinary conditions, while, on the other hand, mental defect showing a retardation of intellectual

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development amounting to three years means failure in life under ordinary conditions.

Psychologists are already making some headway in determining occupational ability by laboratory tests, and this is very suggestive of future possibilities in determining facts of larger prognostic value, by laboratory reactions in the domain of "emotion" and "will."

Of course, the heredity in the case should be taken into consideration, especially when mental defect is found to be slight, or when non-conformity is not associated with intellectual defect or any psychosis.

ABSTRACT OF DISCUSSION

DR. MARY STRONG, Omaha: Before I studied medicine I taught school for about twelve years and had opportunity to observe the various grades of mental defects. Every single one of these children will grow up to be some kind of a degenerate. Alcoholism in one or both parents or the giving of alcohol to children is, of course, a common cause. I have now under my own care quite a number of various grades of degenerates. Most of these children do manual work well, but they are not capable of mental work.

I am attending physician to a rescue home, and at least 25 per cent. of the girls in the home are degenerates. Some are high-grade degenerates. I believe that the prenatal influence of the mother has an influence on the intelligence of the child.

DR. C. F. WAHRER, Fort Madison, Wis.: The early diagnosis of mental deficiency is important. Why? Because this class of persons must ultimately come under municipal supervision, state or national. After we diagnose the cases what shall we do with the patients? Put them into institutions in which they are trained to a semblance of normality so that they can mix with the normal and intermarry and bring down the normal or supernormal to subnormal and keep on producing a race of mental degenerates? Here I believe in the brutal doctrine of "survival of the fittest" and if we, through state or national resources, diagnose these cases and help to educate the children to self-help so that they can get on without the aid of their neighbors, then let us also register them, and when the time comes for certificates of fitness for marriage, and that must mean also fitness for production, we must say that these particular persons cannot marry. If we keep on raising degenerates through propagation of those we already have, we shall eventually have so many that there will not be enough taxable normal persons to keep charge

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of the abnormal. Humanity bids us to be our brother's keeper, but as we can never hope to make normal beings out of defectives, and as we owe it to the sum total of humanity not to overburden the world with such defectives, so we should well ponder these problems, and not indulge ourselves in pseudohumane efforts that will be as the nursing of vipers in our bosoms. Let us see to it that these classes do not multiply and vitiate the purer streams of life.

Dr. A. C. Rogers, Faribault, Minn.: We understand that about 25 per cent. of criminals are mental defectives. Also a large percentage of prostitutes.

The cold-blooded idea of "survival of the fittest" holds good until it comes to our own children. The wisest solution at present, it seems to me, is the segregation of defectives; put them in communities where they can have interests and are absolutely protected from propagating their kind. Possibly some time we may have to do differently, but at present this seems to be the obvious policy. Sterilization has been advocated and has a definite application, but it has greater limitations than most people imagine.

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